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LARKSPUR POISONING OF LIVE STOCK.

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PART I.—INTRODUCTORY.

HISTORICAL SUMMARY AND REVIEW OF LITERATURE.

There is somewhat extensive literature in regard to the larkspurs. In this summary and review only the more important and significant publications are noted, with especial reference to those that treat of the poisoning of domestic animals.

The larkspurs have been known from very ancient times as poisonous and medicinal plants. Under the names σταφὶς ἀγρία of Dioscorides and Hippocrates, ἀγροτέρη σταφὶς of Nicander, *Astaphis agria* or *Staphis* of Pliny, and *Herba pedicularia* of Scribonius Largus, was probably recognized the species *Delphinium staphisagria* L. Under the name *Consolida regalis* were probably included several species. The question of the identity of the species noted by the ancients is discussed in some detail by Huth, 1895, pages 325 and 326.[1]

[1] Full titles of articles referred to in the text are given in the list of literature at the end of the paper.

Pliny speaks of the use of the powdered seeds to destroy parasitic insects on the head and other parts of the body, and this has been its principal use up to the present time, although it has been recommended as a remedy for various ills. As an insecticide the seeds of *Delphinium staphysagria* or "stavesacre" have been much used, but other species have served the same purpose. The leaves, stems, and roots have had little medicinal use, and very little has been published in regard to their poisonous properties besides the investigations on American species. Pliny states that the flowers when ground up serve as a remedy for snake bite. Dioscorides says that the herb paralyzes scorpions when put upon them. Watt, 1890, page 65, says that the root is applied to kill maggots in the wounds of goats. Froggatt, 1900, page 181, recommends larkspur as an insect barrier in gardens. He says that locusts readily eat the leaves and flowers and are killed by them.

Outside of America very little has been published in regard to the poisonous effect of larkspur on the higher animals. Delafond, 1843, page 173, makes the statement that *Delphinium consolida* L. is poisonous to sheep. His evidence does not seem to be extensive, and apparently is based upon the fact that he found sheep dead and, on examination, discovered that they had been eating *Delphinium consolida*. Gerlach, 1845, page 125, says that *Delphinium consolida* has been considered poisonous, but incorrectly, and states that he has fed sheep for several days with the plant and that they ate it readily but received no harm. Dammann, 1886, page 840, quotes Delafond, saying that sheep eat *Delphinium consolida* freely and that when they eat much are poisoned, and states the results of Gerlach. He also quotes Beier, 1845, who tells of horses poisoned by an extract of seeds of *Delphinium staphysagria* in beer. Watt, 1890, page 64, says that the dew from the leaves of *Delphinium brunonianum* Royl falling on grass is said to poison cattle and horses. He also says, 1890, page 69, that the leaves of *Delphinium vestitum* are poisonous to goats. Macgregor, 1908, page 502, gives details of the poisoning of a horse by Delphinium.

From this brief review of the subject it appears that there is little definite evidence that domestic animals in Europe and Asia have been poisoned by larkspurs. Most of the statements are of a general character, no specific instances being given, and they are not based upon personal experiences of the authors. Statements to the effect that animals are poisoned by dew falling from the plants, as in the case of *Delphinium brunonianum*, must be dismissed as purely imaginative. It would seem, therefore, that in Europe and Asia not only is there no loss of domestic animals by larkspur, but also that there are hardly any reliable records of individual cases of poisoning.

It is in North America that practically all the losses of domestic animals from this plant have occurred, and even here the published records are brief and of comparatively recent date. Complaints of losses came, by letter, to the United States Department of Agriculculture many years ago, and newspaper reports of losses have not been uncommon. Philip Miller, in 1760, says of a larkspur, which must be *Delphinium exaltatum* Aiton: "This plant grows naturally in most parts of North America, where, when the cattle happen to feed upon the leaves, it occasions great disorders in them." There seems to have been no other published statement of the poisoning of cattle until the paper by Aven Nelson, 1896, page 79, who said that *Delphinium geyeri* Greene is "frequently greedily eaten by hungry cattle with fatal results, caused by bloating." Earlier, in 1889, Irish, page 25, reported the feeding of cattle upon larkspur with no results. Wilcox, in 1897, published his paper on the poisoning of sheep by larkspur, and this was republished in the Fifteenth Annual Report of the Bureau of Animal Industry, 1898. He says, pages 39 to 43, that from a band of 2,000 yearling lambs, about 50 died and between 500 and 600 showed signs of sickness. Autopsies were made upon the dead animals, and in the stomach contents were found the stems, leaves, and roots of *Delphinium menziesii* D. C.[1] An examination was made of the range over which the sheep had been passing and it was found that the larkspur grew in considerable abundance, and there was evidence that the sheep had been feeding almost exclusively where there was a large quantity of larkspur. Not only that, but it was clear that they had eaten freely of the plant. An examination showed that the plants broke off readily above the root and the inference was that the grazing had been largely of the upper part of the plant, very little of the root having been consumed. After a careful inspection of the other plants upon the range the conclusion was reached that there was no other plant which could be responsible for these cases. Wilcox sums up the results in the following words:

> Thus the post-mortem condition of the sheep, the finding of larkspur in the stomachs of the dead sheep, and the evidence from the field work that the larkspur had been eaten by them seemed to indicate conclusively that the larkspur was the cause of the trouble.

He then gives in some detail the symptoms of larkspur poisoning in sheep, which correspond very closely with the observations of other authors upon larkspur poisoning. In order to make the work more conclusive, extracts of larkspur were made upon the range of Mr. Vestal, at Bigtimber, Mont. The chloroform extract of 25 grams of the dried plant was fed to a lamb, producing symptoms of

[1] This is probably incorrectly determined and should be *Delphinium bicolor*.

poisoning in 30 minutes and death in 2 hours. A second lamb was given, hypodermically, one dram of the chloroform extract, and a third lamb received in a similar manner one dram of benzol extract. Both of these animals showed symptoms of poisoning in 15 minutes, but later recovered after having received, hypodermically, doses of atropine with inhalations of ammonia.

Knowles, in 1897, in the "First Annual Report of the Board of Sheep Commissioners of Montana," speaks of the losses of both cattle and sheep and recommends as remedies ammonia, alcohol, atropine, digitalis, and nux vomica. He says that the most serious losses are among sheep. This article was issued apparently as a circular of the Montana State veterinarian's office in advance of the publication of the report of the sheep commissioners.

Chesnut, in his three publications of 1898, speaks of *Delphinium tricorne* Michx., *D. geyeri* Greene, *D. menziesii* D. C., *D. recurvatum* Greene, *D. scopulorum* Gray, and *D. trolliifolium* Gray as poisonous to stock. Macoun, 1898, states in the Report on the Poison Weed of the Rocky Mountain Foothills that he examined the stomach contents of cattle that had died in the neighborhood of Calgary, making also an investigation of the plants of the region where the animals had died, and came to the conclusion that without doubt the deaths were caused by eating *Delphinium scopulorum* Gray. Willing, 1899, states that a number of sheep are supposed to have died from larkspur poisoning in the Cypress Hills district. In Bulletin No. 2 of the Government of the Northwest Territories, 1900, larkspur is discussed and the experience of Prof. Macoun is referred to, with quotations from Wilcox, 1897.

Wilcox, 1899, discusses the tall larkspur as a poisonous plant for cattle in Montana. He describes the locations in which the plant grows, giving a general description of the plant itself, and states that the principal losses of cattle occur in the spring, after late snowstorms, when the larkspur is the only plant which appears above the snow. He does not think that any very large number of cattle are poisoned in any single year, but that the sum total of the loss is a rather serious matter, and recommends that the cattle be kept away from the larkspur areas, especially after spring snowstorms.

In 1901 was published Chesnut and Wilcox's Stock-Poisoning Plants of Montana. This bulletin discusses in considerable detail *Delphinium glaucum* Wats. and *D. bicolor* Nutt. as poisonous plants, and details are given of the experimental feeding of these plants to rabbits and sheep. A series of experiments was made, using extracts of tall larkspur, identified as *Delphinium glaucum*. These extracts were made in water and alcohol. In one of the experiments the expressed juice of the plant before flowering was fed directly into the stomach of a sheep. Symptoms of poisoning were noticed,

although the animal recovered rather quickly. After expressing the watery material from the plant the alcohol extract of the residue was fed to a sheep. Symptoms of the effect of the alcohol were noted, but by comparison with a check which received the same amount of alcohol, it was decided that some of the symptoms were characteristic of larkspur poisoning. Two other experiments were made in which it was believed by the authors that the antidote used, potassium permanganate, overcame the effect of the poison. The discussion of tall larkspur is summarized as follows, page 73:

The tall larkspur is a plant widely distributed in Montana, occurring, as a rule, in well-defined areas, especially on mountain ranges.

It has for several years been suspected of poisoning cattle, especially after snowstorms in spring and autumn.

Our observations show that the plant is sometimes eaten by cattle with fatal results. Extracts of the leaves of young plants, when fed to rabbits, produce alarming symptoms, and the same was true in one case when fed to sheep.

Experiments on cattle and one sheep indicated that permanganate of potash is an effective antidote when given in the first stages of poisoning.

Cattle should be kept away from patches of larkspur, especially during snowstorms.

The following summary is given of the discussion in regard to purple larkspur, page 80:

The purple larkspur is a plant which is widely distributed in Montana, especially on foothills and mountains, where its deep-blue flowers are conspicuous over wide areas in springtime. For a number of years it has been considered fatal to sheep and occasionally to other stock and this view has been confirmed by our investigations. Sheep are more often poisoned by purple larkspur than are other domestic animals. Our observations during the past few years have shown a striking variation in the appetite of sheep with reference to this point.

Our experiments indicate that both the leaves and roots of young plants are poisonous and that the plant is most dangerous during the early stages of growth before flowering.

The previous experience of one of us has shown that atropine is the best antidote for counteracting the physiological effect of this plant. Permanganate of potash and sulphate of aluminum should be administered as a chemical antidote.

Bessey, 1902, says that there have been serious losses in western Nebraska from *Delphinium nelsonii* Greene, and that the losses occur before the flowering of the plant. Slade, 1903, speaks briefly of Delphinium, the statements apparently being largely compiled from the work of Wilcox. Blankinship, 1903, describes briefly the tall and the low larkspurs. He says that larkspur frequently causes bloat, and gives other symptoms of poisoning, stating that cattle are mainly affected, sheep more rarely. He advises keeping stock away from ranges where low larkspur is abundant, especially during the early spring, and states that it is feasible to dig up the tall larkspur over limited areas.

S. B. Nelson, in 1906, performed a series of experiments, feeding *Delphinium menziesii* D. C. and *D. simplex* Doug. Eight experiments were made with *Delphinium menziesii*, consisting of the direct feeding of both mature and immature plants, and of hypodermic injections of alcoholic and chloroform extracts. As much as 26 pounds of this plant, gathered in full bloom, was fed and apparently 3 pounds and 10 ounces of *Delphinium simplex*. All of these experiments were without results and he reached the definite conclusion that *Delphinium menziesii* is not poisonous to sheep and therefore that they may be allowed to graze where this plant grows in abundance without any fear of loss.

Glover, 1906, gives a somewhat extended description of the larkspurs as poisonous plants. He finds that five species of larkspur are abundant in Colorado—*Delphinium nelsonii* Greene, *D. elongatum* Rydb., *D. geyeri* Greene, *D. barbeyi* Huth, and *D. penardii* Huth. He made an attempt to get exact information from the stockmen of Colorado in regard to their losses and the remedies used, and summarized the results obtained from the circulars sent out. He describes in some detail the appearance of the larkspurs, the symptoms of poisoning, and discusses the best methods of treatment. He says:

> From the reports in other Western States, especially Montana, it would seem that the purple larkspur which is more generally eaten by sheep is the more disastrous of the two. In this State it is quite the reverse. The tall larkspur is more abundant and the major part of the mortality is among cattle.

It would seem from this that Dr. Glover does not question the fact that sheep may be poisoned by eating larkspur. The same thing is indicated by his giving the symptoms of larkspur poisoning in sheep, page 23. He summarizes the conclusions obtained in regard to larkspur poisoning, as follows, page 18:

> First. At least 18 species, and several varieties of larkspur, have been found growing in the State. Four growing in the greatest abundance are known to contain an active poison in sufficient quantities to be dangerous to live stock.
>
> Second. Death is produced as a result of the presence of an active poison, and not from "bloat," as many stockmen have claimed.
>
> Third. The toxic principle of larkspur has not yet been determined for these species, but is probably delphinin and allied alkaloids present in other species that have not been fully studied.
>
> Fourth. The plant loses its toxic qualities as it approaches the flowering season and finally becomes harmless.
>
> Fifth. Two species, because of their abundance, are doing most of the damage, i. e., tall larkspur (*Delphinium elongatum*) and purple larkspur (*Delphinium nelsonii*).
>
> Sixth. Stockmen generally have little knowledge of the identity, poisonous nature, or satisfactory remedy for larkspur.
>
> Seventh. Considering the enormous loss and the fact that larkspur is usually found in circumscribed areas, it would seem feasible, in many localities at least, to undertake its eradication by the grubbing hoe.

Eighth. By avoiding the areas where larkspur abounds during the months of April, May, and June the loss can be reduced to the minimum.

Ninth. In potassium permanganate and atropin sulphate, respectively, we have a chemical and physiological antidote of real practical value. Stimulants are indicated. Tapping should be done with trocar and cannula high up on the left side, after first making slight incision on the skin with a knife. In case of extreme distention this operation should not be delayed. The value of bleeding is questionable. All measures which tend to depress the animal, such as forcible exercise, tobacco, aconite, etc., are positively harmful. If on sloping ground, the head should be turned up the hill.

Crawford, 1907, quotes preceding authors in regard to the effect of larkspur upon stock, but adds nothing to what has been written before. Pammel, 1910, page 44, states that "cattle and sheep are most susceptible, although horses frequently suffer."

Preceding the publication of the present general report on the larkspur investigation, there was issued in 1913 Farmers' Bulletin 531, entitled "Larkspur or Poison Weed," which gave some of the practical results of the work. In 1915 Hall and Yates recapitulate the results of this bulletin, applying them to the larkspurs of California.

It will be seen from the foregoing that up to the time when the detailed experiments of larkspur poisoning were undertaken by the Bureau of Plant Industry, a very definite body of evidence had been accumulated indicating that American larkspurs were poisonous to domestic animals, especially cattle and sheep, causing heavy annual losses in the mountain ranges. There was a fair amount of agreement in the descriptions of the symptoms of poisoning. The remedial measures recommended were very largely those worked out by Wilcox, and by Chestnut and Wilcox in their Montana work. There were, however, several questions with regard to the poisoning which for practical purposes had to be decided. In the published observations and in the statements made by stockmen, the reports were somewhat contradictory with regard to which part of the plant is most poisonous, although there was a general agreement that the principal losses occur in the spring. It seemed necessary to determine at what time of the year and under what conditions these plants are poisonous, to determine whether the tall larkspurs and the low larkspurs are equally poisonous, to describe in somewhat greater detail the symptoms of poisoning and pathological results, and to make further and more detailed experiments upon the possibilities of using remedial measures to lessen the losses. There were also open questions concerning the best method of handling stock so as to prevent poisoning.

It may be noted that practically all accounts of larkspur poisoning of stock in the United States relate to the mountainous regions of the West. As will be seen later in this paper, there is no reason to

think that the eastern species are not poisonous, but conditions of grazing are so different in the East that cattle do not come in contact with the plant to any extent. Recently specific accounts have come to this office of the poisoning of cows by *Delphinium tricorne* in West Virginia.

For convenience of reference, there is given below a list of the species of Delphinium that are said to be poisonous to stock in the United States. This list is compiled from the literature of the subject, from office correspondence, and from personal interviews with stockmen, and no attempt has been made to edit it critically from the standpoint of the systematist. So far as specimens have come to the office of Poisonous Plant Investigations they have been determined by botanists of the Bureau of Plant Industry, but published statements have been taken at their face value.

Delphinium andersonii Gray.
Delphinium barbeyi Huth.
Delphinium bicolor Nutt.
Delphinium californicum T. & G.
Delphinium carolinianum Walt.
Delphinium consolida L.
Delphinium cucullatum A. Nels.
Delphinium elongatum Rydb.
Delphinium exaltatum Ait.
Delphinium geyeri Greene.
Delphinium glaucum Wats.
Delphinium hesperium Gray.
Delphinium macrophyllum Wooton.
Delphinium menziesii D. C., *D. nelsonii* Greene.
Delphinium multiflorum Rydb.
Delphinium occidentale Wats.
Delphinium recurvatum Greene.
Delphinium robustum Rydb.
Delphinium sapellonis Ckll.
Delphinium scaposum Greene.
Delphinium scopulorum Gray.
Delphinium simplex Doug.
Delphinium treleasei Bush.
Delphinium tricorne Michx.
Delphinium trolliifolium Gray.
Delphinium virescens Nutt., *D. penardii* Huth.

THE ALKALOIDS OF DELPHINIUMS.

Most of the laboratory work on the poisonous properties of the Delphiniums has been done in Europe on the seeds of *Delphinium staphisagria*, inasmuch as the seeds of this plant have been used since ancient times as a parasiticide and to some extent for medicinal purposes.

The analysis of the seeds of *Delphinium staphisagria* shows that they contain four alkaloids, namely, delphinin or delphin, delphinoidin, delphisin, and staphisagrin. The chemical composition of these alkaloids has been given somewhat differently by investigators. Marquis, 1877, who claimed to have first obtained the pure alkaloids, gives the formulas as follows:

Delphinin, $C_{22}H_{35}NO_6$.
Delphinoidin, $C_{42}H_{68}N_2O_7$.
Delphisin, $C_{27}H_{46}N_2O_4$.
Staphisagrin, $C_{22}H_{33}NO_5$.

The most characteristic and important alkaloids are delphinin and staphisagrin, and of the two, delphinin has been investigated the more thoroughly and is the more powerful alkaloid. The results

obtained by the various authors who have investigated the physiological action of delphinin have been somewhat contradictory, although the principal symptoms obtained in poisoned animals seem to be quite constant. The cause of some of the discrepancies is probably due to the fact that all the preparations of delphinin used in the various experiments have not been identical. A large variety of animals have been used in the physiological experimentation, including mammals, birds, reptiles, amphibians, and fish, although most of the experiments were performed on frogs and dogs.

Orfila in 1817 gives the following summary of conclusions:

First. That stavesacre is not absorbed, and that its deleterious properties depend on the local irritation it produces and the sympathetic lesion of the nervous system.

Second. That the part soluble in water is most active; so likewise the local effects of its administration are more severe when it is moistened before being applied to the cellular texture.

In 1843 he obtained the following symptoms with delphinin in dogs: For about two hours, nausea and attempts to vomit; then great agitation for some minutes, the dog soon becoming weak and finally lying motionless on its side; slight convulsive movements of the muscles of the legs and lower jaw, followed by death after two or three hours. The organs of sight and hearing remained normal until death. The autopsy showed the mucous membrane of the stomach to be slightly inflamed; the left ventricle contained dark-colored blood, and the lungs were more solid than normal.

Falck and Rörig in 1851 obtained in cats and dogs vomiting, excessive salivation, diarrhea, uneasiness, staggering gait, convulsions, difficult breathing, followed by death from asphyxiation and heart paralysis. The autopsies showed congestion of the mucous membranes which had come in contact with the poison, the heart and great veins gorged with blood, and the lungs covered with ecchymotic spots. Later authors do not vary much in regard to the general symptoms. Van Praag and Turnbull note in addition a diuretic effect.

Cayrade, 1869, states his conclusions as follows:

1. The delphinin acts upon the spinal cord, causing depression and making it lose its excito-motor power.

2. The effects are gradual and are felt from below upward, the reflex power being lost progressively, first in the lower limbs, then in the upper limbs, and, finally, in the head.

3. The voluntary movements continue after the loss of the reflex movements and become incoordinate before their disappearance.

4. The facts observed in the study of normal reflex movements and during the poisoning of the cord by delphinin justify the belief that the nerve cells of the gray matter may lose their power of direct reaction and yet permit the passage of the reflex current.

5. The delphinin seems to act successively and with a paralyzing effect upon the general sensitiveness, the reflex power, the respiration, and the coordination of movements. Its favorite place of predilection is the nervous system and it has no influence on the muscular system.

According to most authors convulsions come on in the later stages of the poisoning, with intervals in which the animal is in a comatose condition. Characteristic of Delphinium poisoning are the muscular tremblings which start in the abdominal muscles and pass over the body. Although most of the authors agree in general on the symptoms and the anatomical lesions exhibited by animals poisoned by delphinin, there is some disagreement as to the way the poison acts in bringing about the observed results. Several authors have compared delphinin to veratrin, and some have compared it to curare, while most of them find that its action is similar to that of its near relative aconitin. It certainly is true that the action of delphinin on experimental animals, as given by most authors, corresponds very closely with the recognized action of aconitin. The principal difference seems to be that delphinin has a direct depressing action on the vasomotor centers of the cord (Boehm and Serck) and that it does not paralyze the heart muscles to any extent (Schiller). Some of the earlier authors attributed the paralysis of Delphinium poisoning to a paralyzing action on the muscles similar to that caused by veratrin, but it has been established that delphinin exerts its essential action on the nervous system rather than directly on the muscles.

Rabuteau and some others advance the theory that the paralysis is due, as in the case of curare poisoning, to the paralysis of the motor end organs rather than to a depression of the nerve centers; while Boehm and Serck describe experiments which show that the preparation of delphinin used by them acted on the motor nerve centers rather than on the end organs.

The chlorid of the alkaloids in the American Delphiniums has been separated by Lohmann and put upon the market by Merck under the name of Delphocurarine, with the idea that it may be used as a drug instead of curare. This has been discussed in some detail by Heyl, 1903.

Authors seem to agree that the slowing of the respiratory movements and the final asphyxiation are due to depression of the respiratory centers in the medulla oblongata and the afferent vagus fibers. Boehm and Serck, 1876, show that death is delayed by using artificial respiration, indicating that asphyxiation is the immediate cause of death rather than the stopping of the heart. They also found that immediately after injections of delphinin, both pulse rate and blood pressure fell, due to the stimulation of the vagus. This is followed by a rise due to paralysis of the vagus through continued action of the poison. If the dose is repeated or if the original

dose is large, the pulse rate and pressure rapidly fall again and the heart stops in diastole.

Hahn, in concluding his article in 1882, gives the following résumé:

The delphin, after having caused a local irritation, which is not very intense in the first stages, manifests its action on the respiration (slowing of the respiratory movements, death by asphyxiation), on the organs of circulation (slowing of the beatings of the heart, lowering of the blood pressure, stopping of the heart in diastole), on the spinal cord (loss of the excito-motor power of the spinal cord, rapidly progressive general anesthesia, convulsions, and paralysis); moreover, the muscles are the seat of intense fibrillar shocks. In its toxic effects delphin then very much resembles the alkaloids of aconite, as one would expect from the botanical relationships; it is distinguished by its energetic action on the nerves supplying the muscles, an action which aconitin does not possess except in a feeble degree.

Keller and Völker in 1913 report the separation from *D. ajacis* of two alkaloids, ajacin and ajaconin. The formula for ajacin is given as $C_{15}H_{21}NO_4H_2O$ and of ajaconin as $C_{17}H_{29}NO_2$. The properties of these alkaloids are given, but apparently no experiments were made to test their effect upon animals.

In 1913 Loy, Heyl, and Hepner made a report of analytical work on Wyoming larkspurs. They isolated an alkaloid in an impure form and made quantitative determinations in *D. nelsonii*, *D. glaucum*, and *D. geyeri*. They state that of these three species, apparently *D. geyeri* is the most poisonous. They find in *D. nelsonii* that the seed contains of the crude alkaloid 1.27, the flower 0.79, the pod 0.60, the root 0.48, the leaf 0.34. In *D. glaucum* they find in the root 1.79, in the flower 0.77 and in the leaf 0.62. In *D. geyeri*, they find in the leaf and stem 1.15 and in the root 0.93.

As is noted later, page 77, the apparent greater toxicity of *D. geyeri* may possibly be explained by the age of the plant.

A review of the laboratory work on the poisonous principles of the Delphiniums brings us to the general conclusion that we have in these plants a poisonous principle similar in its action to that of aconitin. The poison is a local irritant causing strong convulsions in the animals as well as pain and nausea. Its systemic action is on the nervous system, depressing the respiratory and vasomotor centers, and paralyzing the motor centers in the cord. The immediate cause of death, then, is asphyxiation; the heart action also is weak and stops about as soon as respiration ceases.

In the summing up of the work of the field experimentation on the Delphiniums, it will be noted that these symptoms agree quite fully with those noted in animals poisoned by feeding upon the plants at the Mount Carbon station.

LOSSES FROM LARKSPUR POISONING.

It is very difficult to get anything like exact statistical reports of the loses caused by larkspur poisoning. In many localities all cases

of poisoning are attributed to this plant, although the stockmen may have a very indefinite idea of what larkspur really is. In other cases, where they have learned that some other poisonous plant has been responsible for the death of animals, larkspur losses, without any doubt, are overlooked. Generally speaking, however, so far as the reported larkspur poisoning refers to the summer ranges in the mountains, considerable reliance can be put upon the facts presented. This is generally true where the losses refer to cattle rather than to sheep.

The reports of Wilcox, 1897, and Chesnut and Wilcox, 1901, give some details with regard to losses of sheep in Montana, Wilcox stating that out of one band of 2,000 yearling lambs, 102 died. The authors, also, have been told by Mr. L. W. Bailey, of Casper, Wyo., that in the Big Horn region in 1908, 7,000 sheep were lost. Mr. Jeff. Crawford, of Casper, stated that in 1907, in the months of April, May, and June, he lost 23 per cent of his sheep. Both Mr. Bailey and Mr. Crawford supposed that the sheep died from larkspur poisoning. As is indicated eleswhere in this report, however, the authors very much doubt whether larkspur is ever the cause of fatalities in the case of sheep, so that in discussing larkspur losses it is felt that the sheep losses can be ignored.

More complete reports of losses have been made from the State of Colorado than from any other region, largely, without doubt, because the experiment work of the Department of Agriculture upon the larkspurs has been mainly centered in that State. Glover, 1906, estimated that the annual losses among the Colorado cattle herds amounts to $40,000. A few concrete examples collected by the authors will give a more definite idea of what this loss means in individual cases:

Mr. Hartman, of Crystal Creek, Colo., reports that in 1884 or 1885, on the Curecanti, out of 500 head of cattle, 35 died within 5 hours. Mr. Creighton, of Crystal Creek, stated that out of one herd of 3,000, 200 died; and out of another of 5,000, 200 died, while from a herd of 6,000, 196 died. The latter fact was not an estimate, but was carefully tallied by one of the stockmen. In 1908, in Washington Gulch, Gunnison County, Colo., 12 head of cattle were found dead. In the same year in a gulch at the upper part of Red Creek in the same county, 22 head of cattle died between 2 o'clock Saturday, June 27, and 2 o'clock Sunday, June 28. In this case nearly all of the cattle belonged to one man. In District No. 4 of the Uncompahgre National Forest, in the spring of 1909, according to the report of Supervisor Spencer, 100 cattle died. Near Axial, Colo., in 1908, Mr. Iles lost 200 head of cattle. In the same year in an area of six or seven square miles near Axial, 25 head of cattle died out of a total of 800. One man in Del Norte, Colo., was reported by the

forest supervisor as having lost 15 per cent of all his cattle. On the Fishlake National Forest in 1915, it was estimated by the forest supervisor that there was a total loss of cattle amounting to $15,000, one man losing 48 out of a total of 400 head.

An attempt has been made from reports that have been sent in from the various grazing areas to get an idea of the percentage of cattle losses. These percentages can not be considered as very reliable, the estimates made varying from 3 to 7 per cent. A considerable number of the persons reporting make an estimate of 5 per cent. This is a very heavy toll to take of the stockmen, and it is probable that with the exception of the losses from loco poisoning, there is no one cause of loss that draws upon the herds so heavily as larkspur poisoning.

The specific examples which have been given have been largely from Colorado, but losses occur in most of the summer ranges in the mountain regions of the West, and it is probable that the apparently greater losses from Colorado are due in part to the more complete reports and in part, perhaps, to the fact that in Colorado there is a larger extent of valuable summer range than in the other States. The reports of losses in the United States come from all the mountain regions between Mexico and the Canadian line and from the Rocky Mountains on the east to the coast ranges on the west. Similar losses have been reported from the Canadian ranges. The major part of these losses occur in May, June, and early July.

COMMON NAMES OF LARKSPURS.

In Europe a number of common names have been applied to the larkspurs, names derived either from the morphology of the plant or its assumed characteristics. Perhaps the most common name is "stavesacre," a corruption of Staphysagria. In England they are also known as "dolphin flower," "king's consound," "knight's spur," "staggerweed," and "lousewort." In Germany the common names are "Rittersporn," "Lerchen Klaue," and "Horn Kummel." In France, "pieds d'alouette," "herbe Sainte-Athalie," "fleur d'amour," are among the more common names.

In the western United States larkspurs are commonly known as "poison," "poison weed," and "cow poison," while in parts of New Mexico the term "peco" is used. In the mountain ranges of the West the larkspurs are generally known and accurately distinguished by the men who handle stock. Before the plants blossom, however, some confuse Delphinium and Geranium, and more fail to distinguish between Delphinium and Aconitum. The leaves of the aconites resemble the larkspur so closely that, inasmuch as they grow in the same localities, it is not strange that they are not always

recognized as different plants. The flowers of larkspur and aconite are so different, however, that few fail to recognize the difference after flowering.

SPECIES OF DELPHINIUM CONCERNED IN LARKSPUR POISONING.

The classification of the species of Delphinium is in a somewhat unsatisfactory condition, and until a thorough revision of this genus has been made it is hardly possible to speak authoritatively in regard to the distribution of the various species. Generally speaking, we find two great groups, the tall and the low larkspurs. The tall larkspurs embrace the species that are more or less closely related to the old species *Delphinium scopulorum* Gray. The form that has been used in the experimental work in Colorado is known provisionally as *Delphinium barbeyi* Huth, and grows at an elevation of 8,000 feet and higher. *Delphinium robustum* Rydb., with which a single feeding experiment was carried on, is also a tall larkspur. The tall larkspur used in the feeding experiment at the Greycliff station was *Delphinium cucullatum* A. Nels., which is common in the mountains of Montana. The species of low larkspur used at the Mount Carbon station was *Delphinium menziesii* D. C., of which the name *Delphinium nelsonii* Greene, is a synonym, while that fed in Montana was *Delphinium bicolor* Nutt. The tall larkspurs grow throughout the season, maturing in the late summer while the low larkspurs mature and die early in July. Although experimental work has not been carried on by the authors in any other States than Colorado and Montana, there is every reason to think that the plants found in other localities have the same properties and produce the same effects as the larkspurs of Colorado.

From the fact that the low larkspur dies early in July, cases of poisoning from this plant occur mainly in the month of June, and it is commonly thought by the stockmen that the plant ceases to be poisonous when it blossoms; but as shown elsewhere in this report, it is probable that it is poisonous during its whole life. The fact that fewer cases of poisoning occur when the plant is in flower is probably because at that time nutrient grasses are more abundant and the animals eat less of the larkspur. The tall larkspurs are also poisonous early in the season and these poisonous properties, as shown elsewhere, may continue until the maturity of the plant. The cases of poisoning which occur in other States are due to species which correspond in general with the tall and low larkspurs of Colorado.

DELPHINIUM BARBEYI Huth.

Delphinium barbeyi (Pl. I) is a perennial, growing from buds at the apex of a long woody root. The stems are pubescent and more

or less viscid. The leaves are large and deeply cleft into about five segments, and these segments are more or less deeply incised. The leaf segments are oblong or obovate-cuneate. The blue-violet flowers are in a dense terminal raceme, the pedicels being longer than the spurs. The ovaries are bluish.

Delphinium barbeyi has a lower limit of altitude of about 8,000 feet, growing from that point nearly to the timber line. It grows best in damp valleys and canyons, where it may form dense masses. It is found in the mountains of Colorado, Wyoming, and Utah, and perhaps in the adjoining States to the north and south. It starts growth early in the spring and at the Mount Carbon station attains a height of from 1 to 2 feet by the month of June, forming succulent bunches much more prominent than the grass, and doubtless somewhat attractive to grazing animals. The plant grows to a height of from 3 to 7 feet, the blossoms appearing about the 1st of July and the seeds the latter part of the month. The exact time of flowering varies, of course, with the season and the altitude. All vegetation at the Mount Carbon station was from one to two weeks earlier in 1910 than in 1909, and at Kebler Pass, 1,000 feet higher than the station, flowering plants were collected for feeding as late as the middle of August. The seeds are shed very soon after being matured, and the plant begins to dry up, the stems and leaves gradually becoming brown and dry.

Delphinium cucullatum A. Nels.

Delphinium cucullatum (Pl. II, fig. 1) resembles *Delphinium barbeyi* very closely in its habit and occurrence. The stems are glabrous and the leaves divided into three to seven segments. The terminal racemes are closely flowered. The sepals are bluish-white, the petals violet, and the ovaries white. The general appearance of the flowers is bluish-gray, this coloration appearing to be constant for the species. Near the Greycliff station the plants blossomed the last of July.

D. cucullatum is found in Montana, Wyoming, Idaho, and as far south as central Utah.

Delphinium robustum Rydb.

Delphinium robustum is a perennial occurring in the mountains from Montana to New Mexico and grows in the same general way as *Delphinium barbeyi*. The stems are puberulent but not viscid. The leaves are divided into five to seven segments, which are long and twice cleft into linear lobes. It has the same general habits as *Delphinium barbeyi*, but does not confine itself so closely to the canyons and is readily distinguished from *barbeyi* by the form of the leaves.

Delphinium menziesii D. C.

Delphinium menziesii (Pl. II, fig. 2, and Pls. III and XIII) is a perennial, growing from a cluster of small tuberous roots from which the stem is easily detached. The stem is slender, simple, and puberulent. The leaves are deeply cleft into segments which are linear in form. The flowers are deep violet-blue in color, on slender pedicels, and arranged in a loose raceme. There may be as few as four to six flowers, but they are more numerous on thrifty plants growing in favorable locations.

Delphinium menziesii grows at altitudes of from 4,000 to 12,000 feet. It is found on open hillsides and in parks, growing in great abundance. The picture of Pass Creek Park (Pl. III) gives an idea of the number of plants found in that locality. When they were in blossom the surface of Pass Creek Park as seen from a neighboring hill presented a uniform blue appearance. In June, 1908, Supervisor Kreutzer, of the Gunnison National Forest, with the senior author, picked and counted 1,340 of the plants in blossom on a square rod near Crystal Creek, Gunnison County.

Delphinium menziesii is widely distributed, being found from the Rocky Mountains to California and Oregon, and from Alberta to New Mexico. It appears soon after the snow has melted, and at high altitudes the plants may be found growing in immediate proximity to snow banks. It grows to a foot in height and the blossoms appear about the middle of May, the time of blossoming varying with the advancement of the season and the altitude. The seeds, which are formed the last of June, are immediately shed and the plant dies down and disappears. After the first week in July the plant is very rare except at the highest altitudes at which it grows.

Delphinium bicolor Nutt.

Delphinium bicolor is a perennial growing from long fibrous fascicled roots. The stem is glabrous or pubescent, and the leaves deeply cut into linear lobes. The rather stout stem is short, not exceeding 12 or 15 inches in height. The raceme has a few flowers much larger than those of *Delphinium menziesii* and of a deep violet-blue color. It is one of the most beautiful of the American larkspurs.

It grows at a lower altitude than *Delphinium menziesii* and, so far as observed, never in such dense masses. Its range is given as from Washington and Oregon to South Dakota. It is the common low larkspur in Montana, and like *D. menziesii*, blossoms about the middle of May and disappears early in July.

DETECTION OF LARKSPUR SPECIES IN STOMACH CONTENTS.

In connection with these studies cases of poisoning not infrequently occur in which the cause of death can not be determined

FIG. 1.—TALL LARKSPUR (DELPHINIUM BARBEYI HUTH) BEFORE FLOWERING.

FIG. 2.—TALL LARKSPUR (DELPHINIUM BARBEYI HUTH) IN FULL BLOOM.

FIG. 1.—TALL LARKSPUR (DELPHINIUM CUCULLATUM A. NELS).

FIG. 2.—LOW LARKSPUR (DELPHINIUM MENZIESII D. C.).

Pass Creek Park, Colo., with Low Larkspur (Delphinium menziesii) in Blossom.

from the readily available evidence, and recourse must be had to a study of the contents of the rumen. On account of the maceration of the plants most of the material is unrecognizable on macroscopic examination, the leaves especially being almost disintegrated. Frequently, however, stems of grasses and other plants retain their structure sufficiently to show some characteristic features, the fibrovascular bundles in many cases being more or less intact when the looser tissues have been disintegrated.

As the poisoning due to *Delphinium barbeyi* was being investigated, an attempt was made to determine whether the stomachs of the poisoned animals contained this plant, by comparing sections of stems found in the rumen with sections of stems of *Delphinium barbeyi*. In this way it was found possible to determine whether an animal had eaten larkspur, and this method was successfully applied in a number of cases where portions of stomach contents had been preserved in formalin. This work led to the sectioning of stems of other species of Delphinium in order to discover whether it was possible to differentiate between the species by stem sections, especially since in the region where the station was located two species of larkspur occur. This work is here recorded, not in any sense as a complete study of the stem anatomy of the genus, but as a few interesting facts brought out by a comparison of cross sections of stems of a number of species of Delphinium.

In looking up the literature of this genus, no anatomical work was found on the American species. A number of articles have been published both in Europe and America on the anatomy of the Ranunculaceæ as a whole and of some of the other genera, but those dealing with Delphinium in detail are few and are European. In 1885 Albert Meyer published an article on the systematic anatomy of the Ranunculaceæ, in which he grouped the genera according to anatomical characters, and also differentiated many of the species, giving a key based on anatomical characters. His work was on the characters shown by cross-sections of stems. Paul Marié, 1885, published an extensive paper on the histological structure of the Ranunculaceæ. In this work the detailed anatomy of all parts of the plant is described for a number of species in each genus, and the distinguishing characters of the family and of the different genera are discussed. The only article which is devoted solely to the anatomy of Delphinium is that of Lenfant, 1897, on the genus Delphinium in a series of contributions to the anatomy of the Ranunculaceæ. The histological structure of four species (two of which, *ajacis* and *consolida*, have been introduced into the United States) is studied for all parts of the plant and for various stages of growth.

The present work includes the following 29 species of Delphinium: *D. ajacis* L., *D. andersonii* Gray (National Herbarium No. 419245), *D. barbeyi* Huth, *D. bicolor* Nutt., *D. blochmannæ* Greene (National Herbarium No. 2060), *D. californicum* T. & G. (National Herbarium No. 419726), *D. cardinale* Hook (National Herbarium No. 1928), *D. carolinianum* Walt. (National Herbarium No. 442717), *D. consolida*, L., *D. cucullatum* Aven Nelson, *D. decorum* F. & M. (National Herbarium No. 1939), *D. depauperatum* Nutt. (National Herbarium No. 529204), *D. geraniifolium* Rydb. (National Herbarium No. 245524), *D. geyeri* Greene, *D. glaucum* Wats., *D. menziesii* D. C. (National Herbarium No. 333235), *D. nudicaule* T. & G. (National Herbarium No. 612398), *D. occidentale* Wats. (National Herbarium No. 506615), *D. recurvatum* Greene, *D. robustum* Rydb., *D. sapellonis* Ckll., *D. scaposum* Greene, *D. scopulorum* Gray (National Herbarium No. 234530), *D. simplex* Dougl. (National Herbarium No. 226416), *D. tricorne* Michx., *D. trolliifolium* Gray, *D. variegatum* Gray (National Herbarium No. 342458), *D. variegatum apiculatum* Greene (National Herbarium No. 1887), and *D. virescens* Nutt.

These species were used, partly because they are the species which have been met in the field work on poisonous plants, and partly because they were convenient to obtain for comparison. The specimens of *barbeyi* and *menziesii* were from fresh specimens which were fixed and embedded in the field, from specimens preserved in alcohol, and from dried specimens. The sections of *sapellonis* and *cucullatum* were from dried plants sent in from the field. The remaining specimens were from the United States National Herbarium, the Economic Herbarium of the Bureau of Plant Industry, and from the collection of Mr. Ivar Tidestrom. In addition to these species of Delphinium, stem sections were made of two species of Aconitum, for the purpose of comparison, since the two genera are very similar in structure, and since the two occur side by side in the field and both are suspected of poisoning stock.

In preparing the dried herbarium material for sectioning it was treated with 2 per cent sodium hydroxid solution for 24 hours, or until the tissues were softened and swollen, then washed thoroughly in water, and put in a 10 per cent glycerin solution, the glycerin being gradually concentrated through a period of several days. The sections were then cut in pith with a hand microtome, and stained with safranin. Perfect sections are not always obtained by using this method, but for the purpose of the identification of stems in field work it is preferable in most cases to embedding.

Comparison of the different species was based solely on the characters appearing in the cross sections of stems. For each species

cross sections of the main stem of the plant were made without reference to any particular point in the stem. In the case of *Delphinium barbeyi* and *D. menziesii* and *Aconitum bakeri*, sections were made from the subterranean portion of the stem, the petiole, and the peduncle. A photomicrograph was made of a portion of a section of a stem of each species, all the photographs being magnified 65 diameters.

The sections of course showed certain characteristics typical of the Ranunculaceæ, the most noticeable being the form and disposition of the fibrovascular bundles. The bundles are of the closed collateral type and are isolated, being separated by wide medullary rays. The xylem mass has in cross section a somewhat V-shaped appearance, the arms of the V partially inclosing the cambium and phloem. There is no interfascicular cambium. This type of bundle is common to the Ranunculaceæ, but is found almost nowhere else among the dicotyledons (Solereder, 1908, p. 18, and Jeliffe, 1899, p. 339). Another feature of the bundle peculiar to the Ranunculaceæ among dicotyledons is that the phloem consists only of sieve tubes and companion cells, with no phloem parenchyma (Strasburger, 1908, p. 113). These facts in regard to the fibrovascular bundles serve to differentiate the Ranunculaceæ from other dicotyledons, but are also points of resemblance to some of the monocotyledons. Therefore in identifying larkspurs in the stomach contents of poisoned cattle it was necessary to differentiate carefully from some of the grasses when only fragments of the stem could be obtained.

The genus Delphinium has a characteristic stem structure, as shown by cross sections. Vesque, 1881, page 28, says that it is impossible to distinguish anatomically the genera of the Ranunculaceæ, but that certain groups of genera can be recognized, and he places Aconitum and Delphinium in one group. Myer, 1885, page 46, in his key, gives means of distinguishing both Delphinium and Aconitum, the latter being differentiated from Delphinium by the presence of a complete ring of sclerenchyma outside the fibrovascular bundles.

In cross section the external circumference of a Delphinium stem is either approximately circular or approaching an octagonal shape, and the stem is hollow. It is covered externally by a layer of epidermal cells whose outside walls form a thickened cuticle. The epidermis usually bears unicellular hairs of varying shape, size, and number, and is pierced by simple stomata. Beneath the epidermis there is a layer of hypodermal cells similar to those of the epidermis but without thickened walls. Inside the hypodermis there are two to five rows of cortical parenchyma cells, bearing chlorophyll, and arranged loosely with intercellular spaces. In one species it was possible to distinguish an endodermis, but as a rule the endodermis can not be distinguished from the other cells of the pericycle. The

pericycle consists of a ring of sclerenchymatous tissue between the cortex and the phloem portion of the fibrovascular bundles, and is composed of the bast fibers of the bundles and the interfascicular sclerenchyma. The cells of the pericycle have thickened walls, especially in the case of the bast fibers, the cells of which are also smaller than those of the interfascicular sclerenchyma. Inside the pericycle are the phloem and xylem portions of the fibrovascular bundles, the bundles being separated by the medullary rays, which are as wide as the bundles, and the cell-walls of which are sometimes thickened so that they are not distinctly marked off from the pericycle. The medullary rays are continuous with the medullary portion of the stem, in which there is a medullary lacuna of varying size.

The fibrovascular bundles are of the closed collateral type, arranged in a single circle, just inside the cortex. In this description the bast fibers are considered as part of the fibrovascular bundle. The group of bast fibers seen in cross section varies from a wedge shape to a somewhat circular shape, and is usually not sharply defined from the interfascicular portion of the pericycle. It partially incloses the phloem and cambium, while the curved outer border of the xylem partially incloses the cambium on the inner side. The phloem consists of sieve tubes and small companion cells. The cambium is composed of several rows of small thin-walled cells, elongated tangentially, lying in a curved line, with the convexity toward the xylem. Between and surrounding the tubes of the xylem proper is a varying amount of xylem parenchyma.

Classified according to cross sections of stems, the 29 species of Delphinium examined fall into six groups, as follows:

Group 1. *Delphinium barbeyi, D. californicum, D. cucullatum, D. geraniifolium, D. glaucum, D. occidentale, D. robustum, D. sapellonis, D. scopulorum, D. troliifolium.*

Group 2. *Delphinium andersonii, D. bicolor, D. decorum, D. depauperatum, D. menziesii, D. nudicaule, D. tricorne.*

Group 3. *Delphinium blochmannæ, D. cardinale.*

Group 4. *Delphinium carolinianum, D. recurvatum, D. simplex, D. variegatum, D. variegatum apiculatum.*

Group 5. *Delphinium geyeri, D. scaposum, D. virescens.*

Group 6. *Delphinium ajacis, D. consolida.*

These six groups may be combined in three main sections. Section I includes only group 1, which comprises all the species which are commonly known as tall or giant larkspurs. Section II includes groups 2, 3, 4, and 5, and in general represents those species known as low larkspurs. Section III consists of group 6, the European *consolida* group.

Delphinium barbeyi has been taken as the type of group 1. Figure 1, *A*, is a diagram of a cross section of a stem *D. barbeyi*,

with only part of the bundles drawn in; B is a diagram of a typical fibrovascular bundle of group 1. In *Delphinium barbeyi* (Pl. IV,

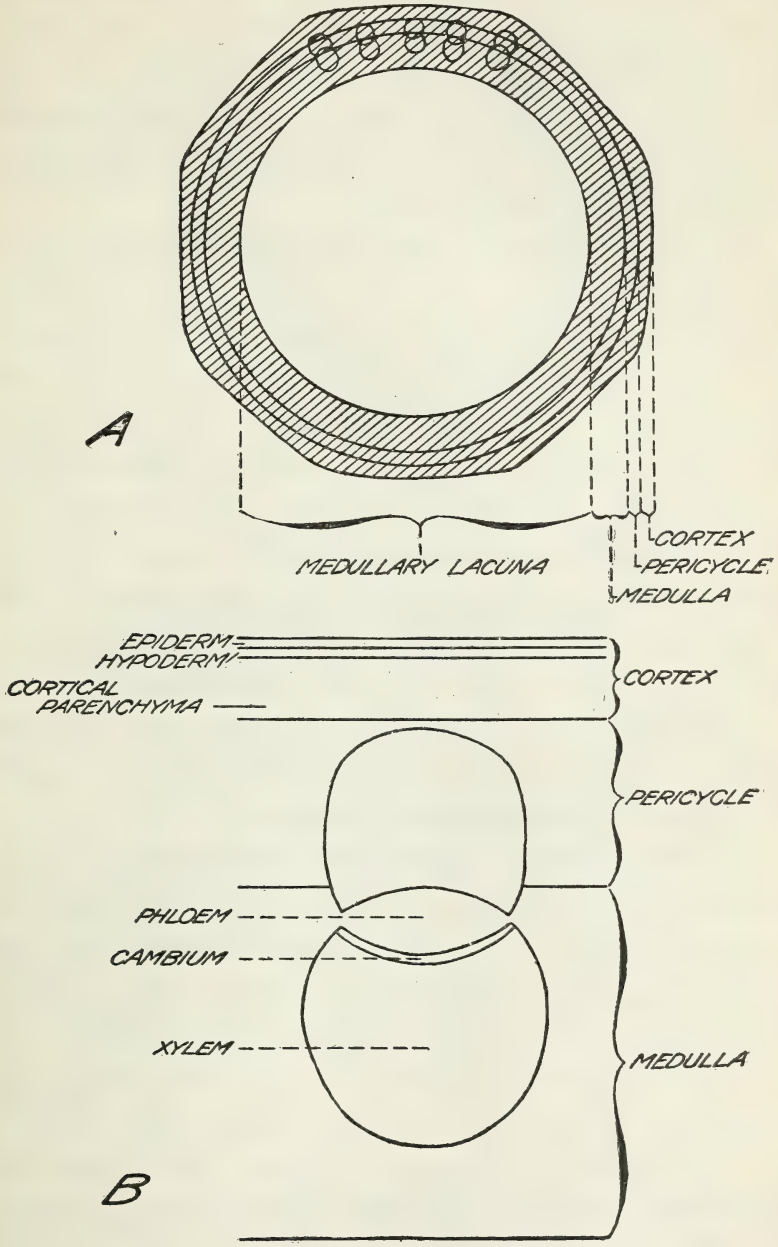

FIG. 1.—*A*. Diagram of cross-section of stem of group 1. *B*. Diagram of fibrovascular bundle of group 1.

fig. 1) the stem is large, with a large medullary lacuna. The outer circumference is roughly octagonal. The bundles are about 32 in

number, and rather small in proportion to the diameter of the stem, those at the angles being a little larger than the others. The cross sections of the xylem and the bast are about the same in size, both being somewhat circular in form. The horns of the bast mass and the xylem mass nearly inclose the lens-shaped phloem. There are only a few rows of xylem parenchyma at the inner end of the xylem. The walls of the cells of the pericycle are not very greatly thickened. The bast fibers of the bundles lying between the angles of the octagon are separated from the cortex by one or two rows of cells continuous with the interfascicular sclerenchyma.

As a type of the second group, *Delphinium menziesii* has been used (Pl. IV, fig. 2). The stem is much smaller than that of *Delphinium barbeyi* and has a medullary lacuna much smaller in proportion to the diameter of the stem. The circumference of the stem is practically circular. The bundles are about 24 in number, of two sizes arranged alternately. The fibrovascular bundle exhibits in cross section a form quite distinct from that of group 1. The bundle is longer and narrower, the bast being wedge-shaped with the larger end situated externally. The phloem portion of the bundle is open laterally, the inner boundary of the bast and the outer line of the xylem being only slightly curved. The xylem proper is small in extent, but there is a large amount of xylem parenchyma extending toward the medullary lacuna.

Group 3 is represented by *Delphinium cardinale* (Pl. V, fig. 1), and in type of stem structure can not be differentiated from group 2. The group 2 type is here exhibited on a larger scale, with a bast larger in amount, and more sharply differentiated from the interfascicular sclerenchyma, and composed of thicker-walled cells, and with a stouter structure all the way through.

In group 4, typified by *Delphinium recurvatum* (Pl. VI, fig. 1), we have a stem structure which may be considered as intermediate between the true low larkspur type of group 2, and the taller forms represented in group 5. The general form of fibrovascular bundle corresponds to that of group 2, but the stem is more compact in structure, the bundles longer and arranged more closely, and the alternate large and small bundle arrangement less prominent.

For the fifth group, *Delphinium geyeri* was used as the type (Pl. V, fig. 2, and fig. 3, *A* and *B*). The medullary lacuna of the stem is very small and the external circumference approaches the octagonal. The bundles are about 30 in number, those at the angles being slightly larger than the others. The cells of all the tissues of the stem are relatively small and numerous. The fibrovascular bundle is similar in the form of cross section to that of group 2, but is larger and much elongated, the bast in particular being very extensive. The bast is oblong to wedge-shaped, and composed of very

small, heavy-walled cells. The xylem proper is small in amount, generally curved at the outer boundary more than is the case in

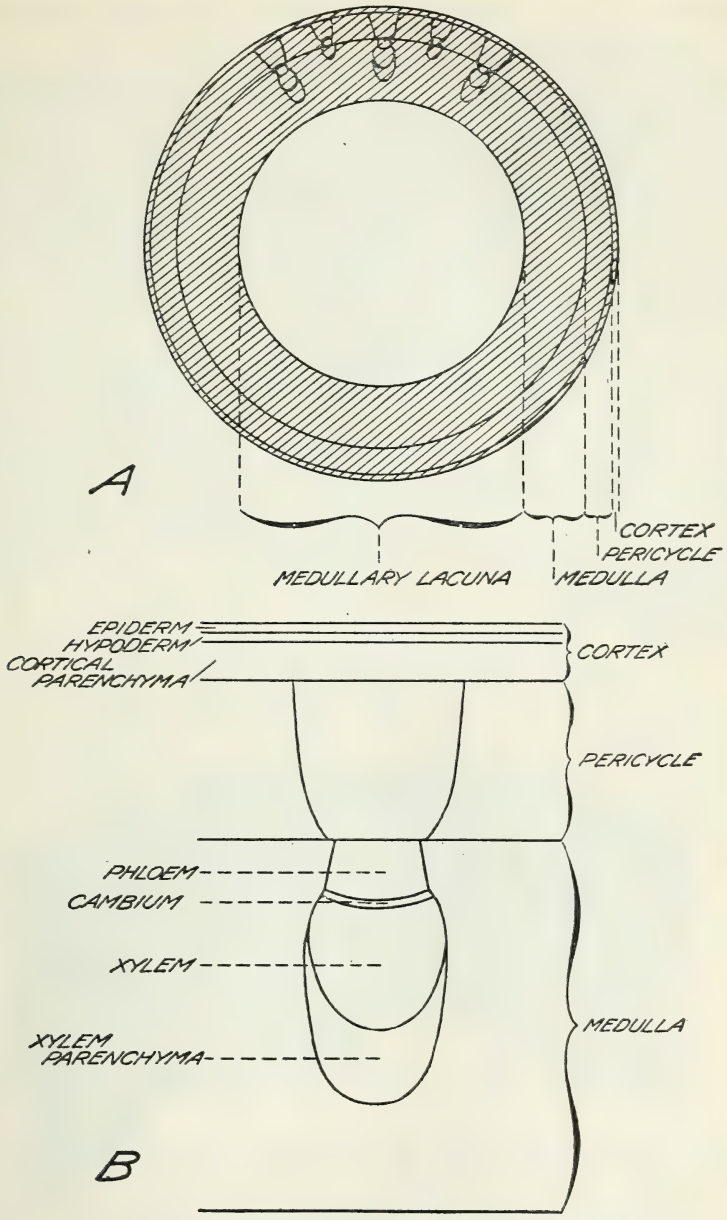

FIG. 2.—*A*. Diagram of cross-section of stem of group 2. *B*. Diagram of fibro-vascular bundle of group 2.

group 2. The xylem parenchyma extends some distance inward from the xylem.

Group 6 is represented by *Delphinium ajacis* (Pl. VI, fig. 2, and fig. 4, *A* and *B*). The stem is circular and has a relatively small medullary lacuna. The bundles are about 46 in number and are of two sizes, the large and small arranged alternately. This is the only group in which it was possible to distinguish a row of endodermal cells. All the cell walls are much thickened, which is a distinguishing characteristic of this group. The shape of the fibrovascular bundles is quite characteristic. The bast is wedge-shaped, composed of cells whose walls are so thickened that the lumen is reduced almost to a point. The phloem is small and completely inclosed by the bast and xylem. The xylem mass is larger than the bast, elongated, and includes a large amount of xylem parenchyma.

Delphinium consolida is similar to *D. ajacis*, but the bundles are less numerous, the cell walls in the pericycle are thickened still further, and part of the cells of the cortical parenchyma have thickened walls.

Any of the species which were examined could be quite easily placed in one of the above groups, but within the groups the work thus far done has not revealed sufficiently characteristic differences in stem structure to make identification of species possible. Vesque, 1881, page 29, says that while it is impossible to distinguish genera by anatomical characters, it is easy to distinguish species, but he uses different characters to differentiate the species, such as the structure of the petiole, the development of palisade cells, and the distribution of stomata in the leaf. On the other hand, the present work is based on stem characters, which serve to differentiate between genera in the family Ranunculaceæ, and in this case between groups of species in the genus, but not between individual species. An exception to this is group 6, of which we have only two species in America, and these two can be distinguished by the anatomy of the stem. These two are European species which have been introduced into the United States, and are described anatomically by Lenfant (1897, pp. 26–27, Pl. VII) and Marié (1885, pp. 117–118, Pl. VI). The specimens of *ajacis* and *consolida* from the National Herbarium which were examined had evidently been misnamed, one for the other, as was discovered by comparing cross sections of the stems with the descriptions and figures of Marié and Lenfant.

Sections were also made of two species of Aconitum, *A. bakeri* Greene (Pl. VI, fig. 3; and fig. 5, *A* and *B*) and an unidentified species from California, in order to compare them with and to differentiate them from the tall larkspurs. The cross section of the stem shows a structure similar to that of the tall larkspurs, but it can be easily distinguished by the lack of a medullary lacuna, and by the complete

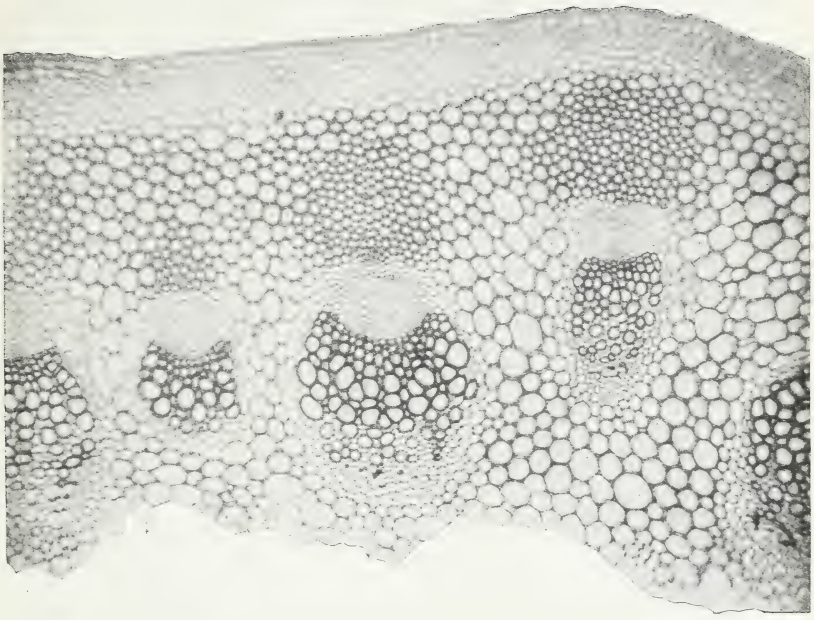

FIG. 1.—CROSS SECTION OF STEM OF DELPHINIUM BARBEYI.

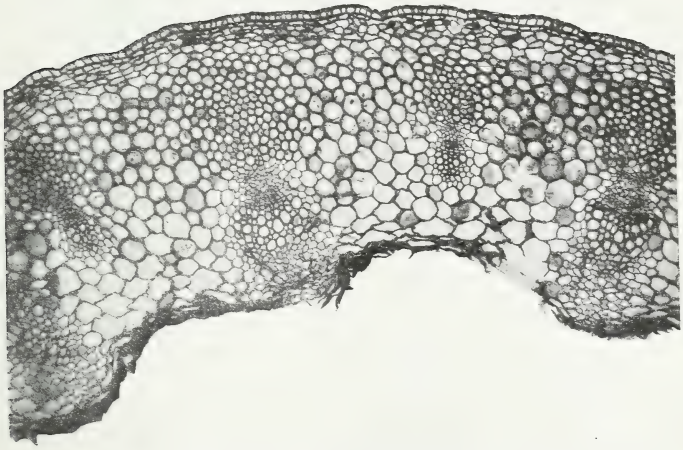

FIG. 2.—CROSS SECTION OF STEM OF DELPHINIUM MENZIESII.

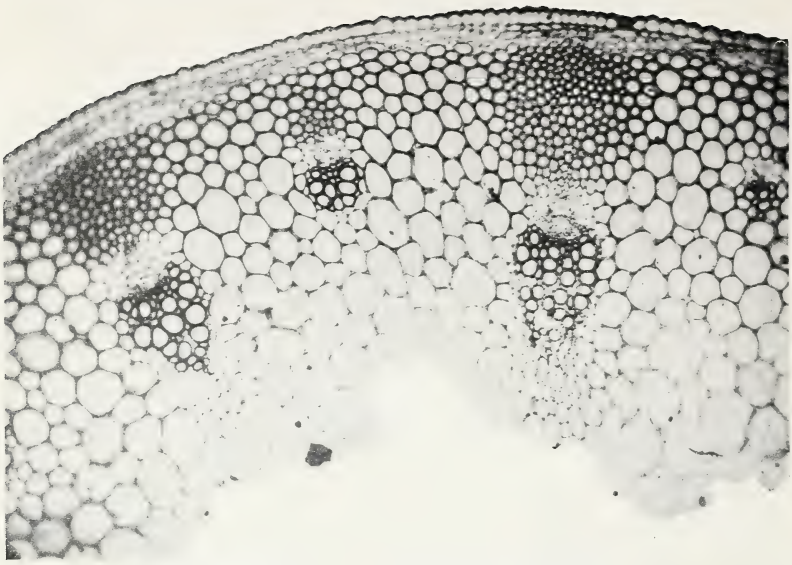

FIG. 1.—CROSS SECTION OF STEM OF DELPHINIUM CARDINALE.

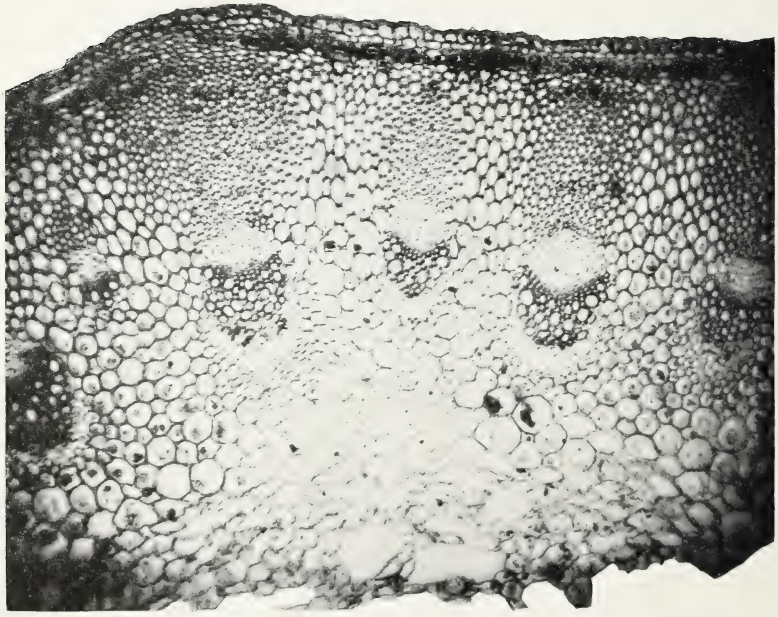

FIG. 2.—CROSS SECTION OF STEM OF DELPHINIUM GEYERI.

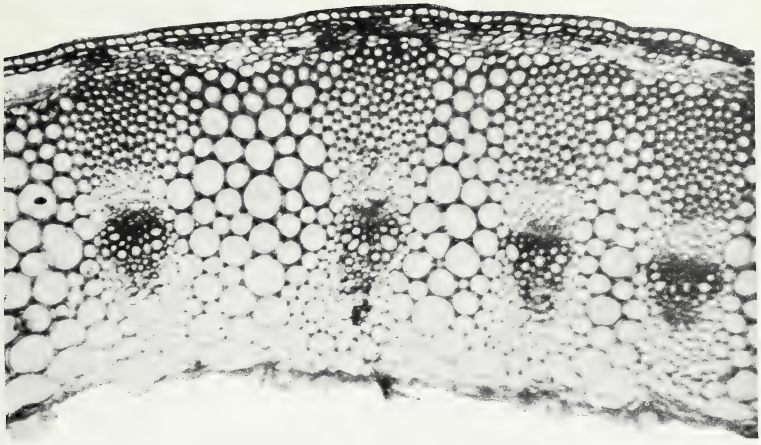

FIG. 1.—CROSS SECTION OF STEM OF DELPHINIUM RECURVATUM.

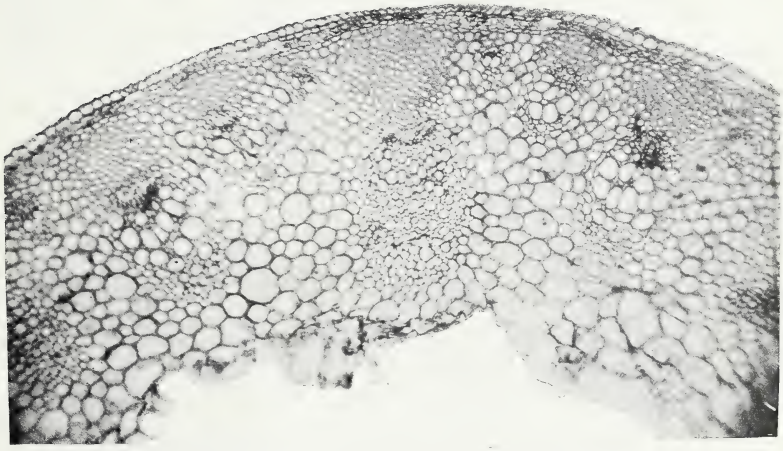

FIG. 2.—CROSS SECTION OF STEM OF DELPHINIUM AJACIS.

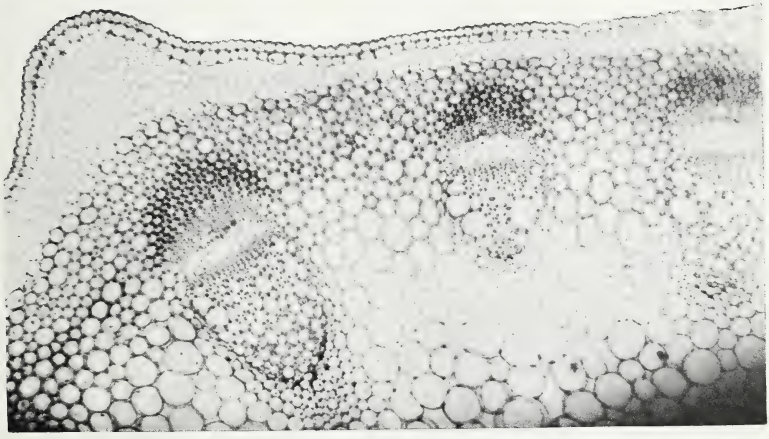

FIG. 3.—CROSS SECTION OF STEM OF ACONITUM BAKERI.

FIG. 1.—STATION AT MOUNT CARBON, COLO.

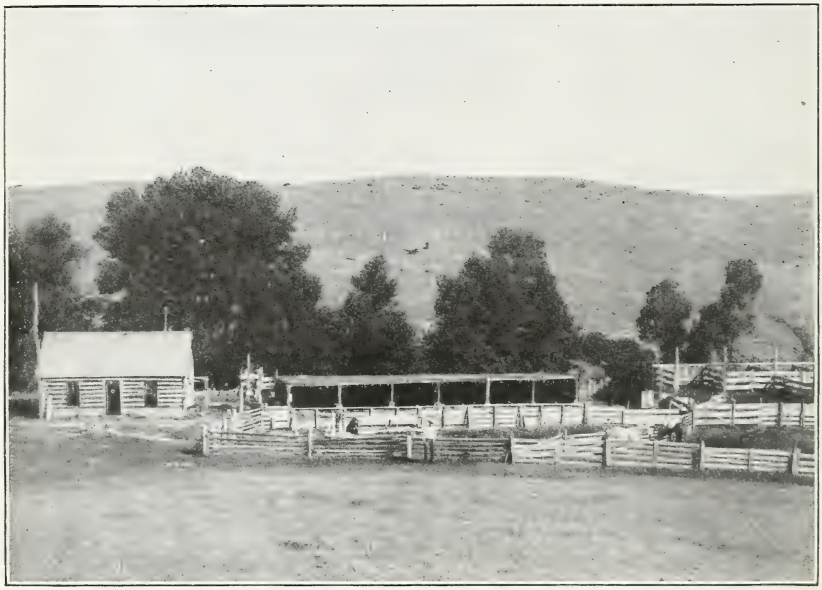

FIG. 2.—STATION AT GREYCLIFF, MONT.

ring of sclerenchyma outside the bast fibers. As is shown in the diagram (fig. 5), the circumference of the stem is circular, with the

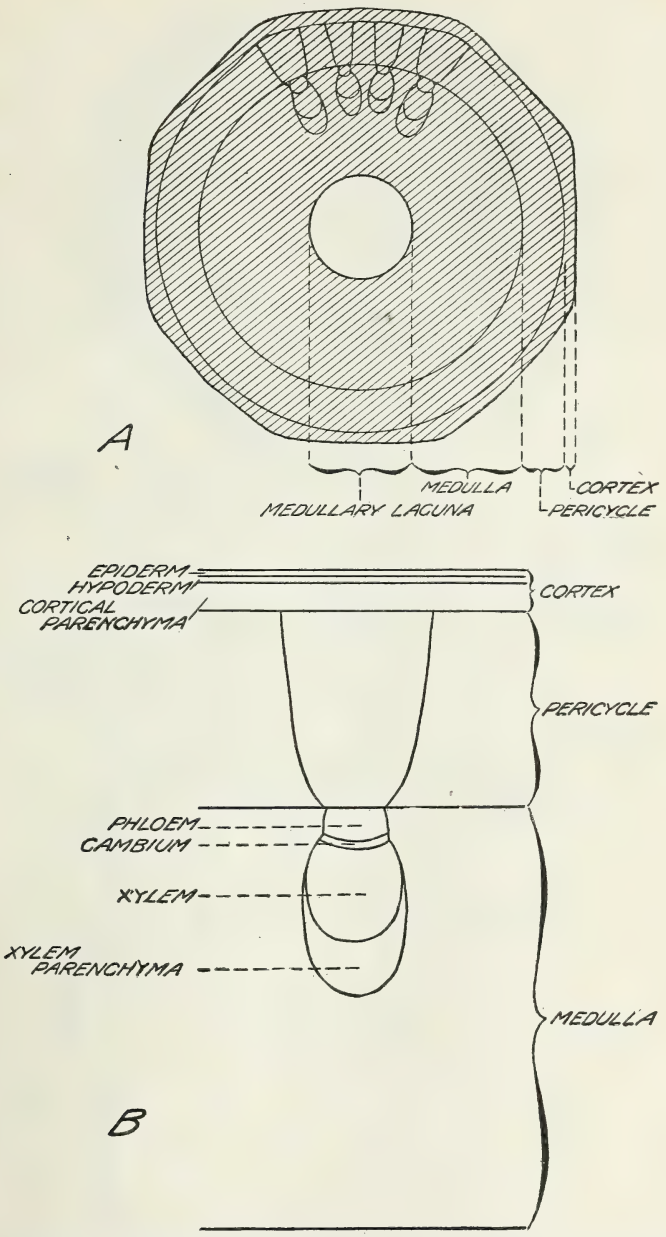

FIG. 3.—*A*. Diagram of cross-section of stem of group 5. *B*. Diagram of fibro-vascular bundle of group 5.

exception that at two points the cortex is thickened. The bundles are of about the same size, and about 30 in number arranged in a single

circle. The pericycle is similar to that of Delphinium, but is distinguished by the fact that there are several layers of thick-walled

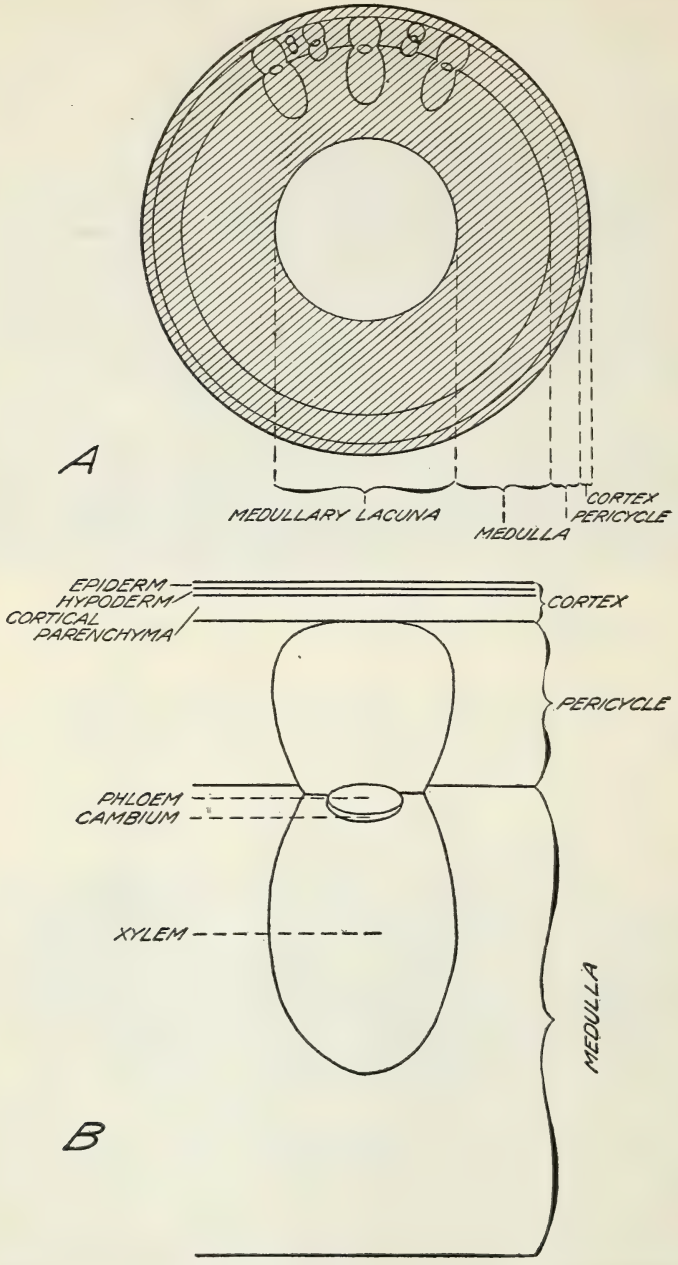

FIG. 4.—*A*. Diagram of cross-section of stem of group 6. *B*. Diagram of fibro-vascular bundle of group 6

cells continuous with the interfascicular sclerenchyma, separating the bast from the cortex. The cross section of the fibrovascular

bundle is similar in size and shape to that of the Delphinium group 1. The bast is smaller and crescent-shaped, while the xylem is long and

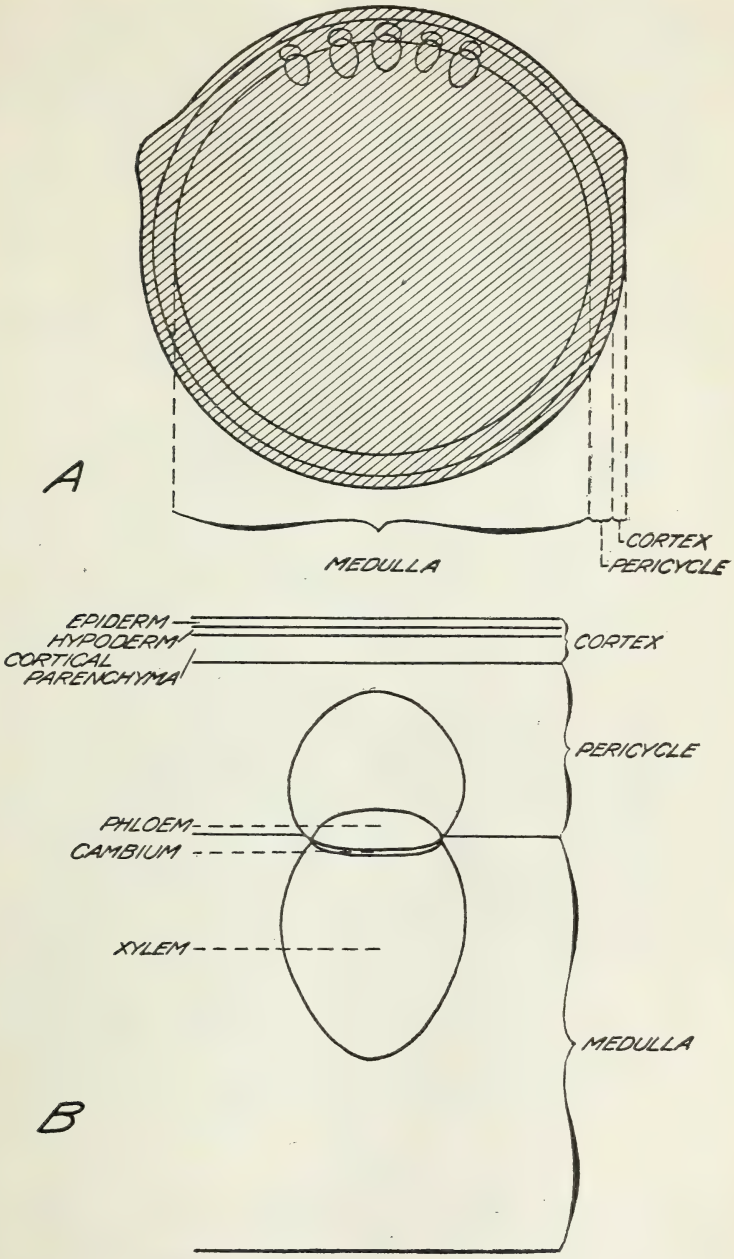

FIG. 5.—*A*. Diagram of cross-section of stem of Aconitum. *B*. Diagram of fibro-vascular bundle of Aconitum.

pointed. The outer border of the xylem is only slightly curved and does not inclose the phloem.

As a result of the study of the stem structure of 30 species of Delphinium and 2 species of Aconitum it has been found possible, by an examination of cross sections of the stems, to distinguish between Delphinium and Aconitum and between six groups of species in the genus Delphinium. This has been put to practical use in the examination of the contents of the rumen of poisoned cattle, by which means it has been possible to determine whether the animal had eaten Delphinium, and to which group of species the plant eaten belonged.

PART II.—EXPERIMENTAL WORK.

THE STATION AT MOUNT CARBON, COLO.

The station for the detailed study of larkspur poisoning was located four miles north of Mount Carbon village, in Gunnison County, Colo. (Pl. VII, fig. 1). Through cooperation with the Forest Service, a ranger's station, including a cabin, barn, corrals, and pastures, was provided for the experimental work. This station was in the Ohio Creek Valley at an elevation of about 9,000 feet, in a region where *Delphinium barbeyi* and *Delphinium menziesii* were extremely abundant. In this region, also, losses which are attributed to larkspur occur every year to a greater or less extent, and in some years the losses have been very heavy. This station was selected, too, because it was a favorable location from which studies could be made upon a number of other plants supposed to be poisonous. It was intended, however, that the principal experimental work should be upon these two species of larkspur. The station was equipped with the necessary laboratory facilities, and arrangements were made for cattle and horses for experimental purposes, the work being inaugurated on June 10, 1909, and continuing through that summer until October 1. In 1910 and 1911 it was resumed about the middle of May, and continued until nearly the 1st of October. During these seasons experimental work was conducted upon cattle, horses, and sheep. Acknowledgment should be made to the Forest Service not only for the assistance rendered by equipping the station, but for the continual help of the officers of the Service during the progress of the experimental work. It is desired also to acknowledge the assistance rendered by the stockmen who had cattle upon the Castle Creek and Anthracite ranges. Through the courtesy of these men a large number of cattle were loaned for the experimental work, and thus much material assistance was rendered. While the experimental work was going on the force kept in close touch with the men controlling the cattle upon the ranges, and one or more members of the station force accompanied the stockmen during the time the cattle were driven from the Castle Creek range to the Anthracite range,

in order to be present at the times when larkspur poisoning was deemed most likely to occur. The location of the station was most favorable, not only because of the abundance of larkspurs in the immediate vicinity, but because it was located in the immediate neighborhood of the summer ranges of the cattle, so that a most intimate knowledge of range conditions could be gained.

THE STATION AT GREYCLIFF, MONT.

In 1912 and 1913 the field experimental work in poisonous plants was carried on at Greycliff, Mont. (Pl. VII, fig. 2). An old sheep-shearing plant was loaned for the purpose by the owner, Ole Birkeland, and the necessary repairs were provided by the Forest Service, including fitting up the house for use as office, laboratory, and dining hall, necessary repairs to the barn, and construction of fences and corrals.

While experimental work was to be undertaken on a number of poisonous plants, this location was considered especially favorable for the study of the effects of feeding *Delphinium cucullatum* and *Delphinium bicolor*. The main industry in this region is sheep grazing, and it was considered an especially favorable point to study the effect of the Montana species of larkspur on sheep. Here, as in Colorado, the stockmen of the neighborhood showed most helpful interest in the work and assisted materially by loaning sheep and cattle for experimental work.

EXPERIMENTAL FEEDING OF DELPHINIUM BARBEYI TO CATTLE IN 1909.

In 1909, 42 experiments were conducted of feeding *Delphinium barbeyi* to cattle on 26 different animals. Table I gives a summarized statement of these feeding experiments. The work was not commenced until the last of June and definite results were not obtained until the last of July. Of these 42 cases 22 were poisoned.

As the season progressed it was evident that larger quantities of the plants were necessary to produce toxic effects than had been supposed at the beginning of the experiments, and this fact doubtless explains the failure to produce poisoning in the earlier experiments. The summarized results in regard to symptoms and treatment are given later in this paper. Following are a few typical cases given in some detail.

Case 92.

This case was interesting as being the first one in which there were definite symptoms of poisoning. Case 92 was a cow weighing about 990 pounds which had been used for experimental purposes with *Delphinium menziesii* without any effect. On June 30 she ate 30 pounds of leaves and stems of *Delphinium barbeyi*. On the

TABLE I.—*Summary of feeding experiments upon cattle with Delphinium barbeyi, 1909.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*						*Pounds.*	
84	740	7.5	June 28–29	Leaves and stems				10.1	Near station.
92	990	30	June 30	do	Not known	Potassium permanganate	Recovery	30	Do.
113	750	26.5	July 2–3	do				35.3	Do.
81	1,230	2	July 8–9	do				1.6	Do.
112	610	6	July 10–11	Leaves, stems, and flowers				9.8	Do.
91	980	17.7	July 17	do				18	Do.
92	990	16.5	July 25–27	do				16.8	Do.
605	450	22.5	July 30–31	Leaves, stems, flowers, and seed.	1 hour	Potassium permanganate	Recovery	50	Do.
605	450	12	Aug. 1	do	35 minutes		do	26.6	Do.
605	450	9.5	do	do	12 hours	Caffein and potassium permanganate.	do	21.1	Do.
97	500	35	Aug. 1–3	do	Death		Death	70	Do.
603	550	34	Aug. 2–4	Leaves, stems, and seed	2½ hours	Atropin, caffein, and potassium permanganate.	Recovery	61.8	Do.
602	450	25	Aug. 5–6	Flowers, seed, and seed stems.	3½ hours	Atropin, caffein, and paunched	do	55.5	Do.
606	450	28	do	Leaves and stems				62.2	Do.
107	420	9	Aug. 7–10	Seed and seed stems				21	Do.
108	430	14.75	do	Leaves and stems				34.3	Do.
98	460	14.75	Aug. 10–11	Flowers				32.1	Kebler Pass.
82	960	63.75	Aug. 10–12	Leaves and stems	45 minutes	Atropin, caffein, and paunched	Recovery	66.4	Kebler Pass and near station.
117	620	32.25	Aug. 13	Leaves, stems, flowers, and seed.	13 hours	Atropin, caffein, and potassium permanganate.	do	52	Kebler Pass.
113	750	23	Aug. 13–14	Seed and seed stems				30.7	Near station.
112	610	38	do	Leaves and stems				62.3	Kebler Pass.
92	990	27.5	do	Seed and seed stems				27.7	Do.
115	610	63.5	Aug. 17–19	Leaves, stems, flowers, and seed.	2½ hours	Potassium permanganate	Recovery	104	Do.
604	450	11.75 72.75	Aug. 17–21	Seed and seed stems Leaves and stems	1⅓ hours	Caffein	do	187.7	Near station. Kebler Pass.
608	700	113.25	do	Leaves, stems, flowers, and seed.	40 minutes		do	161.8	Kebler Pass.
603	550	95.75	Aug. 19–22	Leaves and stems	2 hours		do	174	Do.
112	610	34.25	Aug. 19–20	Seed and seed stems	30 minutes	Atropin	do	56.1	Do.
108	430	22.75	Aug. 24–25	Leaves and stems	Sick but not down.		do	52.9	Do.
98	460	48	do	Leaves, stems, and seed	2 hours		do	104.3	Do.

107	420	50.5	Aug. 24–27	do	4 hours	Magnesium sulphate	do	120.2	Do.
81	1,230	161.25	Aug. 27–Sept. 4	do	Death		Death	131.1	Kebler Pass and near Salt Lick.
605	450	29.5	Aug. 28–29	Seeds and seed stems	5 hours	Caffein	Recovery	65.5	Do.
606	450	195	Aug. 28–Sept. 4	Leaves and stems		Magnesium sulphate		433.3	Do.
113	750	32.75	Sept. 2–4	Seed and seed stems				43.6	Kebler Pass in part.
113	750	60.5	Sept. 9–10	do	2 hours	Atropin	Recovery	10	Kebler Pass.
602	450	388.25	Sept. 9–16	Leaves, stems, and seed		Magnesium sulphate		862.8	Do.
608	700	280.5	do	do				400.7	Near station and near Salt Lick.
112	610	79.5	Sept. 16–17	do	Death		Death	130.3	Kebler Pass.
84	740	292.25	Sept. 16–23	do				394.8	Salt Lick and above Coke Ovens.
92	990	316	Sept. 18–25	do				319.2	West side of Mt. Carbon.
98	460	357.25	do	do		Magnesium sulphate		776.6	Kebler Pass.
115	610	132	Sept. 25–28	do				216.4	Do.

morning of July 1 it was noticed that she staggered as she walked, her hind legs appearing stiff. She gave evidence also of some abdominal pain. This peculiar stiffness in gait continued through the day of July 1 and was still noticeable on the morning of July 2. No other pronounced symptoms of poisoning were noticed.

CASE 605.

Case 605 was a yearling heifer loaned for experimental purposes by Mr. J. H. Eilebrecht. She was estimated to weigh about 450 pounds.

During July 30 and 31 she received 35 pounds of *Delphinium barbeyi*, the material including stems, leaves, and some flowers and seeds. This material was chopped up and mixed with chopped hay in order that the animal might eat it more readily. She was fed at 5 p. m. on July 31 and was apparently entirely normal. At 5.30 it was noticed that she appeared somewhat weak upon her hind legs when forced to walk about the corral. She soon fell, her fore legs giving away first, and she was unable to get up. She moaned as though in pain. Several times she tried to get up but apparently did not have sufficient strength. Her pulse at this time was 60, her temperature 102.2° F. There was no evidence of bloating. At 6 p. m. respiration was 70 and rather irregular. The pulse was slower than when observed before. At 6.11 she suddenly got upon her feet and walked away. She was weak and staggered but otherwise seemed all right. No further symptoms were noticed during that evening.

It was noticed that during this illness she urinated rather freely. She appeared well on the morning of August 1 and the feeding was resumed, giving her as before stems and leaves of *Delphinium barbeyi* with some flowers and seed, the material being cut up and fed with hay. During the forenoon she ate 12 pounds of this material. At 1.15 p. m. while walking about in the corral she suddenly fell and was unable to rise. The pulse was 68, respiration 68 and somewhat irregular. She was constipated and moaned as though in pain. At 1.25 her temperature was 102.3. At 1.30 she suddenly got upon her feet, ran around the corral, and fell down again. At 1.45 her pulse was 60 and respiration 45. At 1.50 she got upon her feet. She stumbled as she attempted to rise, but did not go down again. When started up she stumbled and fell upon her knees, but was able again to get upon her feet. As she stood, the abdominal muscles contracted as if she were in great pain and there was also spasmodic twitching of the muscles of the shoulders.

She remained on her feet after this time and as she appeared normal the feeding was resumed at 3 p. m. She ate 9½ pounds. At

FIG. 1.—CASE 603 AT 4.45 P. M., AUGUST 21, 1909.

FIG. 2.—CASE 603 AT 4.54 P. M., AUGUST 21, 1909.

FIG. 3.—CASE 603 AT 4.54¼ P. M., AUGUST 21, 1909.

FIG. 4.—CASE 603 AT 4.54½ P. M., AUGUST 21, 1909.

FIG. 5.—CASE 603 AT 4.54¾ P. M., AUGUST 21, 1909.

FIG. 6.—CASE 603 AT 4.58 P. M., AUGUST 21, 1909.

FIG. 1.—CASE 603 AT 4.58½ P. M., AUGUST 21, 1909.

FIG. 2.—CASE 603 AT 4.59 P. M., AUGUST 21, 1909.

FIG. 3.—CASE 603 AT 5.15 P. M., AUGUST 21, 1909.

FIG. 4.—CASE 603 AT 5.15¼ P. M., AUGUST 21, 1909.

FIG. 5.—CASE 603 AT 5.15¾ P. M., AUGUST 21, 1909.

FIG. 6.—CASE 603 AT 5.16 P. M., AUGUST 21, 1909.

FIG. 1.—CASE 117 SHOWING HIND LEGS BRACED APART IN THE EFFORT TO REMAIN STANDING.

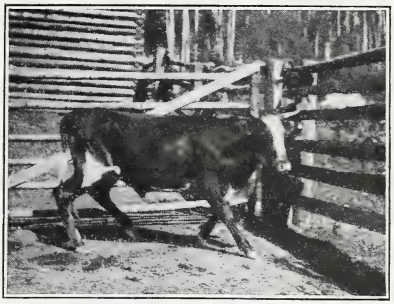

FIG. 2.—CASE 117, AUGUST 15, STAGGERING.

FIG. 3.—CASE 117, AUGUST 15, REMAINING ON ITS FEET WITH GREAT DIFFICULTY.

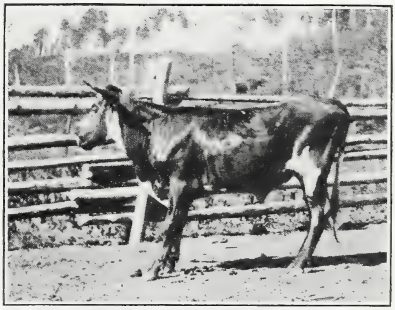

FIG. 4.—CASE 117, AUGUST 15, IN THE ACT OF BACKING IN THE MANNER CHARACTERISTIC OF LARKSPUR POISONING.

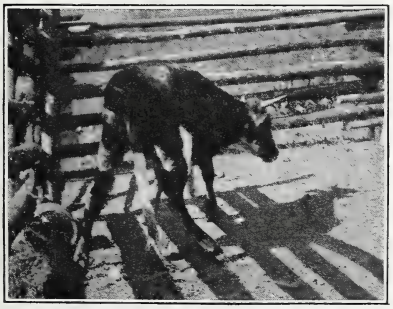

FIG. 5.—CASE 117, AUGUST 15, JUST BEFORE FALLING.

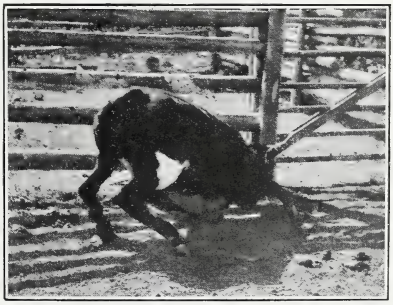

FIG. 6.—CASE 117, AUGUST 15, FALLING IN THE MANNER TYPICAL OF LARKSPUR POISONING.

FIG. 1.—CASE 117, AUGUST 15, JUST AFTER AN ATTEMPT TO RISE.

FIG. 2.—CASE 117, AUGUST 15, 9.10 A. M., ATTEMPTING TO RISE.

FIG. 3.—CASE 117, AUGUST 15, 9.35 A. M., AGAIN ATTEMPTING TO RISE.

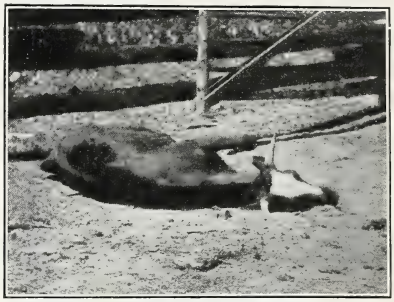

FIG. 4.—CASE 117, AUGUST 15, 10 A. M., UNABLE TO MOVE.

FIG. 5.—CASE 117, AUGUST 15, 12.05 P. M.

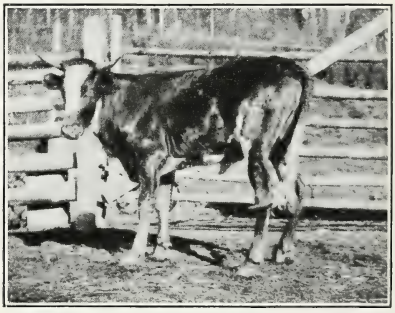

FIG. 6.—CASE 117, AUGUST 17, AFTER RECOVERING FROM POISONING.

6.50 p. m. she was found down again and unable to rise. She was moaning as if in pain. At 7.20 her pulse was 65, and at 10.45 it was 60 and somewhat stronger. She remained down during the night unable to rise, but at 6.45 a. m., on the following morning, she got upon her feet, moved about and although she fell, was able to rise again. A little later, however, she stumbled and fell and could not rise, but at 8.15 a. m. she was again upon her feet and eating as though hungry. At 10.15 a. m. she appeared quite well, with the exception of some weakness, and was turned back into the pasture with the other animals.

During the first of this series of illnesses she was given a drench of potassium permanganate, the treatment being repeated in the evening. There seemed to be no reason, however, to think that this had any definite effect. She was also given hypodermically an injection of 25 grains of caffein sodio-benzoate at 10.45 in the evening. There was no evidence that this produced any effect. This case was particularly interesting because of the successive illnesses produced by renewed feeding of the *Delphinium barbeyi*.

CASE 603.

Case 603 was a yearling heifer, weighing about 550 pounds, which was loaned to the station for experimental purposes by Mr. O. E. Wiseman. From August 2 to August 4 she received 34 pounds of *Delphinium barbeyi*, including stems, leaves, flowers, and buds. This was mixed with hay and corn chop in order that it might be eaten with greater readiness. No effects were noticed until the afternoon of August 4. She was apparently well at 4.30. At 6.50 she was found lying flat on her side and at first was supposed to be dead. She was breathing, however, and soon kicked a little. A dose of 1 grain of atropin was administered subcutaneously. She was raised up so that she lay upon her belly with her head off the ground. In this position she held her head around by her side as if in pain. At 6.55 respiration was 24 and the pulse between 75 and 80 and weak. At this time she was given a drench of potassium permanganate. At 7.03 respiration was 23 and temperature 101.2° F. At 7.15 a hypodermic injection of 30 grains of caffein sodio-benzoate was given. At 7.30 the temperature was 101.3° F. At 7.45 she attempted to get upon her feet but was unable. At 8.20 respiration was 22, pulse about 90 and not very strong. At 9.10 she was upon her feet and from this time showed no further symptoms of poisoning.

She was brought into the corrals for further feeding on August 18. Between August 19 and August 22 she ate 95.75 pounds of *Delphinium barbeyi*, the material including stems and leaves. At 4.35 on August 22 she was found lying with her head turned to the right of the body. She got up, staggered about and fell, but lay with head

erect. At 4.54 she began to walk about uneasily, staggering, and finally fell, going down upon her forefeet first, with her head extended upon the ground.

Plate VIII, figure 1, shows her attitude while lying down at 4.45, and figures 2, 3, 4, and 5 show successive attitudes taken by the animal during the minute from 4.54 to 4.55; figure 2 shows her with arched back and lowered head, in the attitude she took while staggering about the corral; figure 3, taken immediately after, shows very nearly the same attitude; while figure 4 shows her after coming down upon her fore legs, with head extended upon the ground in an attitude which is very characteristic of animals poisoned by larkspur; figure 6 shows her again upon her feet at 4.58. At 4.58 she commenced to stagger, and was upon the ground at 5 o'clock. Plate VIII, figure 6, and Plate IX, figures 1 and 2, show her successive attitudes in this process. She arose again at 5.14, but fell almost immediately. Plate IX, figures 3, 4, 5, and 6, show her attitudes at this time, and it will be noticed that they are comparable with the two preceding series. These four pictures were taken within less than a minute. At 5.26 she was again upon her feet, but at 5.30 commenced to stagger, backing around the corral in a way that was found to be characteristic of larkspur-poisoning cases. She attempted to defecate, moving her head up and down as if in great distress, and then fell down again. She was upon her feet again at 5.44, but at 5.53 fell. Her respiration at this time was 30. At 6 o'clock she was again upon her feet, but moved her head up and down, stepping about uneasily, backing as before. She staggered somewhat, reminding one very much of the actions of a drunken man. At 6.04 she lay down, but at 6.07 got up with no apparent difficulty and began picking up hay in the corrals. At 6.15 she showed uneasiness, moving her head up and down. Then she lay down again. During this latter time she went down voluntarily and was evidently improving, for during the earlier stages of the poisoning she was entirely unable to get upon her feet after falling. At 7.15 she seemed normal, and no further symptoms of poisoning were noticed.

During this case of poisoning there was an interval of two hours from the time the animal first fell until the time when she was able to remain standing.

CASE 117.

Case 117 was a steer weighing about 620 pounds. On August 13 he was fed stems, leaves, and flowers, and a few seed pods of *Delphinium barbeyi*, receiving 32.25 pounds.

On the morning of August 14, at 8.30, it was noticed that he was acting in a somewhat abnormal manner. When walking he kept

upon his feet with difficulty, his legs being too weak to hold him up. Some of the time when standing he would tremble, and at times he would place his legs wide apart as if to keep from falling over. This was particularly noticeable as he walked down hill. Sometimes in walking he would stagger to one side or the other. It was noticed that he urinated quite frequently but the quantity was not great. At 10.30 he seemed to be stronger upon his legs and no marked change was noticed during the rest of the day. Several times he was found lying down but was able to get up without much difficulty.

As showing his weakness it was noticed that when he swung his head around to brush off flies the movement would cause a loss of balance so that he would stagger and almost fall.

Plate X, figures 1, 2, 3, and 4, show some of the attitudes assumed by him during the day. When first seen on the morning of August 15, between 6 and 7 o'clock, his condition did not seem to be changed from that noticed on the preceding day. He was upon his feet and moving about a little. At 8.15 he seemed much weaker. He was down and made no effort to get up. Even with assistance, he was unable to raise the fore part of the body. Plate X, figures 5 and 6, show his attitude at this time; in figure 5 he was trying to hold himself upon his feet while in figure 6 he was falling again. At 8.25 he was given a drench of potassium permanganate. His heart action was very weak at this time and it was with great difficulty that his pulse could be detected. Respiration seemed normal, although his breathing apparently caused pain. At 8.30 he was given subcutaneously 1 grain of atropin dissolved in camphor water. A little after this he tried to get up but was unable. He could not get his forequarters off the ground, but did succeed in moving himself around. Plate XI, figure 1, shows him just as he had fallen back after an attempt to get upon his feet. During the rest of the day he made several attempts to get up but was generally unable to raise his hindquarters from the ground. It was evident that he was in constant pain and this forced him to attempt to change his position. At 9.55 a. m. his pulse was about 95, his respiration 36. The pupils were very much dilated from this time on, probably from the influence of the atropin. There were spasmodic contractions of the abdominal muscles.

Plate XI, figure 2, shows the animal attempting to get up at 9.10; figure 3 shows him at 9.35 when he was attempting without success to get up. The abdominal pain was apparently very severe. At 10.30 he was given subcutaneously 25 grains of caffein sodiobenzoate. At 10.40 his temperature was 102.4° F. Plate XI, figure 4, shows his attitude at 10 a. m. and figure 5 shows him at 12.05, noon. At 2.45 he seemed weaker than at any preceding time and the pulse was hardly perceptible. He was given 1 grain of atropin in cam-

phor water. At 3.25 the pulse was fairly strong. At 4.25 he very nearly succeeded in getting upon his feet. The muscles of the shoulders and flanks were trembling much of the time.

As he was much constipated, feces being discharged only once during the day, he was given at 6 p. m. 12 ounces of Epsom salt as a drench. At 9.10 p. m. he appeared very much brighter than at any time during the day. Trembling was not so pronounced and the pain was less. He breathed normally, held his head from the ground and took notice of what was passing around him. He was not seen again until the morning of August 16. At 6.45 a. m. on August 16 he got up, ate a little hay and drank water. During the forenoon of August 16 he lay down most of the time but occasionally got up and walked from place to place. The improvement continued during the afternoon and night. He still staggered when walking and remained upon his feet only a few minutes, but could get up and down at will. On the morning of August 17 there was still some trembling of the surface muscles of the shoulders. Plate XI, figure 6, was taken at 7.25 a. m. on August 17 when he appeared fairly normal.

He was driven back into the pasture still showing weakness, trembling, and staggering when hurried, but after this his recovery was rapid and complete.

EXPERIMENTAL FEEDING OF DELPHINIUM BARBEYI TO CATTLE IN 1910.

The experimental feeding of *Delphinium barbeyi* in 1909 had indicated somewhat clearly the symptoms of poisoning and the dosage so that the work of 1910 was largely directed to experiments with various remedies. The discussion of these remedies is taken up later in this paper. Table II gives a summary of the experimental feeding of *Delphinium barbeyi* to cattle during this second summer. Forty-three feeding experiments were conducted on 24 different animals. Following is a detailed description of some of the more typical cases.

Case 612.

Case 612 was a yearling heifer loaned for experimental purposes and weighing about 500 pounds. From July 2 to July 5 she received 76.5 pounds of *Delphinium barbeyi*, including leaves, stems, and flowers. At 4.15 p. m. on July 5, as the animal had apparently felt no effect from the feeding, an attempt was made to run her about the corral. After being run about a few times she began to tremble, her legs giving out, and she fell and was unable to rise. Respiration was 60 and irregular and the pulse 160 and weak. At 4.20 she fell over upon her side, the surface muscles contracting spasmodically. At 4.24 the pulse was 100 and rather weak. At 4.27 she was given

subcutaneously one-half grain of atropin. At 4.29 the pulse was between 95 and 100, respiration 46 and slower and deeper than when noticed before. At 4.38 respiration was 60 and irregular. At 4.40 the pulse was 75 to 80. At 4.51 respiration was 40 and the pulse 94. At 5.01 she suddenly got up without any apparent effort and walked the length of the corral. She stood for a moment, trembling violently, then fell, going over upon her left side. At 5.30 an attempt was made to get her upon her feet, when she began to vomit. She was held up for about ten minutes, until it was evident that there was no regurgitated material in the lungs or trachea. At 5.55 she attempted to get upon her feet, but was unable. At 6.10 she was given a hypodermic injection of one-fourth grain atropin, and at 6.30 she was given hypodermically 10 cubic centimeters of undiluted whisky. At 6.45 she lay with head extended, eyes partly closed, lips apart, muscles of the flanks twitching, with rapid breathing, and was apparently about to die. At 6.55 she was given a second dose of 10 cubic centimeters of undiluted whisky. At 7.10 her head was raised and she was able to keep it erect. At this time she attempted to get up and made another attempt at 7.12. At 7.22 she got up, went the length of the corral and walked about nervously. There was still some twitching of the muscles of the body. From this time on she seemed to improve in condition and showed no other symptoms of poisoning. There seemed to be no doubt that in this case the injection of whisky had bridged over a period of weakness which otherwise might have ended fatally.

Case 118.

Case 118 was a yearling steer born August 9, 1909, whose estimated weight was 300 pounds. He received July 7, 18.25 pounds of *Delphinium barbeyi* including stems, leaves, and blossoms. This was given in three feedings, one at 9.15 a. m., one at 9.40 a. m., and one at 2.40 p. m. At 3.55 he was found down and unable to get up, apparently from weakness. At 4 p. m. the pulse was 70 and rather weak, respiration 72. At 4.09 respiration was 100 and pulse 75. Saliva was running from his mouth. At 4.28 the pulse was 60. At 5.01 there were a few spasmodic contractions of the legs, but nothing that could be considered as convulsions. During these spasmodic contractions he went over on his left side and remained there. Respiration was 54. During this time he had made several unsuccessful attempts to rise. There was some belching of gas from the stomach.

Two subcutaneous injections of atropin were given, the quantity given being one-half grain in all. The respiration became more and more shallow and soon stopped entirely. An attempt was made to stimulate it by inhalation of ammonia, but it was unsuccessful.

TABLE II.—*Summary of feeding experiments upon cattle with Delphinium barbeyi, 1910.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of live weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*						*Pounds.*	
98	650	19.75	May 30	Leaves and stems	Death	Barium chlorid, caffein, and strychnin.	Death	30.4	Near station.
107	600	90	June 11–17	do	do		do	150	Do.
613	450	38.5	June 29–July 1	do	do	Physostigmin, pilocarpin and strychnin.	do	85.5	Do.
612	500	76.5	July 2–5	Leaves, stems, and flowers	3 hours	Atropin and whisky	Recovery	153	Do.
82	1,000	101.5	July 6–11	do				101.5	Do.
118	300	18.25	July 7	Flowers	Death	Atropin and alcohol	Death	60.6	Do.
610	510	20	July 13	Leaves, stems, and flowers	9 hours	Physostigmin, pilocarpin and strychnin, and whisky.	Recovery	40	Do.
626	450	30	July 19	Leaves, stems, flowers, and few seeds.	15 hours and 16 minutes.	Physostigmin, pilocarpin, and strychnin.	do	66.7	Do.
627	500	20	do	Leaves, stems, flowers, and seed.				40	Do.
617	400	28	July 24	Leaves, stems, and seed	25 minutes	Physostigmin pilocarpin, and strychnin.	Recovery	70	Do.
622	500	32	July 24–28	Leaves, stems, and flowers				64	Do.
628	500	46	July 26–28	Leaves, stems, flowers, and seed.				92	Do.
620	500	42	July 29–30	Leaves, stems, and seed	2 hours and 20 minutes.	Physostigmin, pilocarpin, and strychnin.	Recovery	84	Do.
624	500	103.5	July 29–Aug. 1	Leaves, stems, flowers, and seed.	16 minutes		do	207	Do.
621	500	31	Aug. 2	Leaves and stems	41 minutes		do	62	Do.
614	500	45	Aug. 2–3	Leaves, stems, and seed	47 minutes	Physostigmin, pilocarpin, and strychnin.	do	90	Do.
609	500	27	Aug. 3–4	Seed and seed stems	Death		Death	54	Do.
618	500	35	Aug. 4–5	Leaves and stems	27 minutes first day; 30 minutes second day.	Physostigmin, pilocarpin, and strychnin.	Recovery	70	Do.
121	300	50	Aug. 4–6	Leaves, stems, and seed	1 hour and 9 minutes.	do	do	167	Do.
625	600	168.5	Aug. 4–8	Leaves, stems, flowers, and seed.	17 minutes	do	do	280.8	Kebler Pass.
610	500	45.5	Aug. 6–7	Leaves and stems	14 minutes	do	do	91	Near station.
612	500	25.5	do	Seed and seed stems	40 minutes	do	do	51	Do.

628	500	55.5	Aug. 8–11	Leaves, stems, and seed	20 minutes first day; 1½ hours second day.	Physostigmin, pilocarpin and strychnin, and whisky.	do	111	Do.
622	500	24	do	Seed and seed stems				48	Do.
627	500	130	Aug. 8–14	Leaves and stems				260	Do.
619	400	101.5	Aug. 10–14	Leaves, stems, flowers, and seed.				253.7	Kebler Pass.
82	1,000	15.5	Aug. 12–15	Seed and seed stems				15.5	Near station.
626	450	156.5	Aug. 12–17	Leaves, stems, and seed				347.8	Do.
617	400	78	Aug. 15–18	Seed and seed stems				195	Salt Lick and Kebler Pass.
620	500	128.5	Aug. 15–19	Leaves and stems				257	Salt Lick.
621	500	157	do	Leaves, stems, and seed				314	Kebler Pass.
622	500	116.5	Aug. 19–23	do				233	Salt Lick.
618	500	44	Aug. 20–23	Seed and seed stems	20 minutes		Recovery	88	Kebler Pass.
625	600	134.5	do	Leaves and stems				224	Salt Lick.
624	500	254.5	Aug. 20–25	Leaves, stems, seed, and few flowers.				509	Kebler Pass.
121	300	29.5	Aug. 24–25	Seed and seed stems	1 hour	Physostigmin, pilocarpin, and strychnin.	Recovery	983	Do.
614	500	354.5	Aug. 24–30	Leaves, stems, and seed				709	Salt Lick.
619	400	26	Aug. 26–28	Seed and seed stems				65	Kebler Pass.
627	500	271.5	Aug. 26–31	Leaves and stems				543	Do.
621	500	291	Sept. 1–7	Leaves, stems, and seed				582	Salt Lick.
628	500	275.5	do	do				551	Kebler Pass.
115	825	78	Sept. 9–13	do				94.5	Salt Lick.
120	250	198	Sept. 9–16	do				792	Kebler Pass.

Fifty cubic centimeters of alcohol was given subcutaneously about the time respiration stopped, but this was evidently too late. The pulse could be felt for about three minutes after respiration had stopped.

An autopsy was made on the morning of July 8. The heart was found to be in diastole with petechiæ upon its walls. The mucous membranes of the larynx and trachea were inflamed and the lungs congested. The walls of the first stomach were congested near the esophageal opening. The walls of the second and third stomach were strongly congested at the cardiac end. The duodenum was congested, the jejunum slightly congested. The ileum was slightly congested throughout its length. There was congestion in the upper part of the cecum. The walls of the rectum near the anus were extruded and inflamed. The kidneys were congested. It was noticeable in this animal that while there was mucus in the trachea and bronchi there had been no actual vomiting.

Case 610.

Case 610 was a yearling heifer weighing about 500 pounds which was loaned by the Castle Creek stockmen. She was fed leaves, stems, and flowers of *Delphinium barbeyi* on July 13, being fed at 9, 9.30, and 10 a. m., eating altogether 20 pounds. At 11.40 she became uneasy and soon went down, and by the time the observer could obtain assistance from the laboratory she was found on her left side, flat upon the ground.

She was immediately given a subcutaneous injection of physostigmin salicylate three-fourths grain, pilocarpin hydrochlorid 1½ grains, and strychnin sulphate one-half grain. At 11.45 respiration was 80 and pulse 64. A picture was taken at 11.49, which shows her lying flat upon the ground (Pl. XII, fig. 1). At this time there was some trembling and some salivation and she was kicking about as though in pain. At 11.45 the pulse was 76, respiration 60 and shallow. At 12.11 the pulse was 75. At 12 o'clock a small amount of feces was passed and more at 12.12. There was a further passage at 12.35. From 12 until about 12.30 considerable gas was expelled from the stomach. At 12.30 she was able to raise herself upon her belly. At 12.35 the pulse was 72. It was noticed that there was considerable secretion during this time from the lachrymal glands. By 1.40 considerable gas had accumulated in the rumen, and as she did not seem to be able to relieve herself by expelling it per os, the trocar was thrust into the rumen. This relieved the pressure and the breathing became easier. The animal lay at this time with her head around to her side in the position shown in Plate XII, figure 2.

From 12.30 on it was noticed that she perspired quite freely. This was probably due to the effect of the remedy pilocarpin. At 2

FIG. 1.—CASE 610 AT 11.49 A. M., JULY 13.

FIG. 2.—CASE 610 AT 11.49½ A. M., JULY 13.

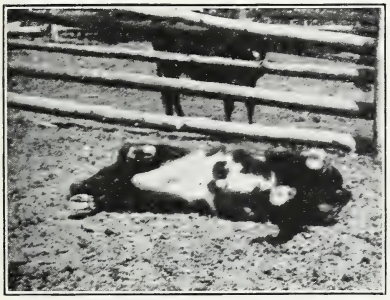

FIG. 3.—CASE 612 AT 1.18 P. M., AUGUST 7.

FIG. 4.—CASE 612 AT 1.30 P. M., AUGUST 7.

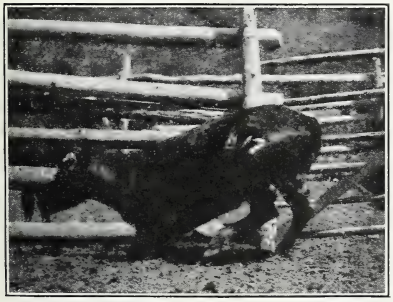

FIG. 5.—CASE 612 AT 1.37 P. M., AUGUST 7.

FIG. 6.—CASE 612 AT 1.47 P. M., AUGUST 7.

FIG. 1.—CASE 82 AT 3.20 P. M.

FIG. 2.—CASE 82 AT 3.24 P. M.

FIG. 3.—CASE 82 AT 3.27 P. M.

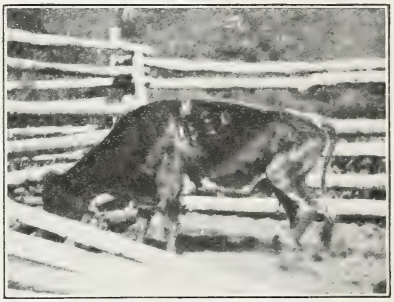

FIG. 4.—CASE 82 AT 3.56 P. M.

FIG. 5.—CASE 82 AT 3.56 P. M., AFTER FALLING.

FIG. 6.—CASE 82 AT 3.59 P. M.

o'clock her respiration was 85, deeper and much more regular than before the gas was allowed to escape from the stomach. At 4.06 the pulse was 80 and apparently weaker, respiration 44. At 4.15 as she seemed to be growing weaker she was given a hypodermic injection of 20 cubic centimeters of whisky. At 4.20 respiration was 40. At 4.25 the pulse was 100 and stronger. While, during the afternoon she had seemed stupid, paying very little attention even to the flies which were around her in great numbers, at 4.52 she became sufficiently lively to attempt to get rid of the flies. There was still some twitching of the muscles of the flanks. At 5.43 the pulse was 86 and respiration 28. At 6.40 respiration was 24. She continued down until 8.03 when she was able to get upon her feet. At 8.06 she arched her back with her hind feet apart and trembled all over. She fell down, going over on her side. The pulse was 90 and weak, the respiration seemed normal. At 8.33 she was able to get up again. She had urinated very little and apparently there had been very little urination for a considerable time before her illness. She was also very much constipated. During the night of July 13 considerable urine was passed and some feces. On the morning of July 14 she was still weak and was kept in the corrals until July 15, when she was turned out as recovered.

Case 612.

Case 612 was brought in for further experimental work on August 1. During August 6 and the forenoon of August 7 she received 25.5 pounds of seeds and seed stems of *Delphinium barbeyi*. At 1.05 p. m. August 7 she was found lying down, but when approached walked away apparently in good condition. At 1.07 her back was arched, she began to tremble, backing around the corral in an uneasy manner, and soon fell, going down upon the forelegs and lying upon the belly. At 1.10 when disturbed there was some muscular twitching of the shoulders. She remained upon her feet until 1.18, when she began to tremble and went down. She lay upon her right side flat upon the ground. Plate XII, figure 3, shows her position at this time. She was rolled over and placed with head erect. At 1.23 her pulse was 80 and weak, respiration 92, and fairly regular. At 1.26 she was given hypodermically physostigmin salicylate, 1 grain; pilocarpin hydrochlorid, 2 grains; and strychnin sulphate, one-half grain.

Plate XII, figure 4, shows her position at 1.30. She had made several unsuccessful attempts to get upon her feet, but at 1.37 was able to get up. Plate XII, figure 5, shows her in the act of rising. She walked across the corral but at 1.38 stumbled and fell again, going over upon her side. At 1.23 respiration was 143. She was

expelling some gas from the stomach. At 1.42 the pulse was 120. At 1.46 the pulse was 104. At 1.47 she raised herself without much effort. Plate XII, figure 6, shows her at this time. At 1.52 she was trembling, her back was arched, and she was stepping about uneasily. There was considerable salivation, and there was and had been for some time dribbling of urine. At 1.55 the trembling was very much decreased. She walked with a stiff gait and at 2.04 seemed to be over the attack. No further symptoms were noted.

EXPERIMENTAL FEEDING OF DELPHINIUM BARBEYI TO CATTLE IN 1911.

Because *Delphinium menziesii* disappears about the first of July, the station work in the early part of the seasons of 1909 and 1910 was very largely concentrated on this plant, and most of the work on *Delphinium barbeyi* was done after the plant was in blossom. As the season in 1911 was about two weeks later than in 1910, *Delphinium barbeyi* in the middle of July in 1911 was in about the same stage of development as at the first of July in 1910. In addition to confirming the work of the preceding seasons on symptoms and remedies, especial attention was paid to the poisonous effects of the plant in its early stages. Two experiments were made of feeding the dried plant, as it was desirable to determine whether the plant lost its poisonous properties by drying.

Twenty-six feeding experiments were conducted on 22 different animals, and the greater poisonous effect of feeding the larkspur within a short period of time was much more clearly brought out than in the preceding seasons.

The experimental work with remedies made it possible to determine quite definitely the quantities of physostigmin, pilocarpin, and strychnin which could be used to the best advantage.

Table III shows the results of the feeding in a summarized form and they are discussed later in the paper. None of the cases are given in detail, since the feeding experiments were conducted in the same manner as in the preceding years and the general results were the same.

EXPERIMENTAL FEEDING OF DELPHINIUM MENZIESII TO CATTLE IN 1909.

During the season of 1909, nine experiments were made of feeding *Delphinium menziesii*, the experiments commencing on June 24 and continuing until July 25. Part of the material used was collected around the station, and was to a large extent mature, the plant being in flower and in some cases containing seeds; the remainder was obtained at Kebler Pass, and consisted of small plants before flowering. The whole plants, including roots, stems, and flowers, were fed to some animals, while in other cases only the tops were fed, and in still others the roots ground up with grain.

It is commonly believed by stockmen that the root of this plant is the most poisonous, and it is generally supposed that the plant produces more cases of poisoning after a rain, because at that time the ground is soft and the animals can pull up the plant by the roots and thus get the part in which the poison is supposed to be concentrated.

Table IV gives a summary of these experimental feedings.

Experiments were made by feeding the roots alone, the animals used being Nos. 92 and 117. Number 92 in two days ate a quantity equivalent to 2.04 pounds per 1,000 pounds of weight, while No. 117 in one day ate 2.1 pounds per 1,000 pounds. The greatest quantity fed at any time was to No. 115, which between July 10 and July 12 received 100.7 pounds of tops, seeds, and flowers per 1,000 pounds of weight. The greatest quantity of the whole plant that was fed, including not only tops but roots, was given to No. 97, which received on July 25 21.2 pounds per 1,000 pounds of weight. No. 91 received 5 pounds on July 2 and 3, and again on July 16 received 21.2 pounds. In none of the cases of feeding *Delphinium menziesii* was there any evidence of toxic effect, although the plant was fed at different stages, part of it before flowering, part after flowering, and even after seed had commenced to form, and attempts were made to find out whether one part of the plant was more poisonous than another.

If it were particularly poisonous it seemed that the feeding in a single day of 21.2 pounds per 1,000 pounds of weight would have produced some effect. It is true, however, that animals upon the range, when hungry, will sometimes eat enormous quantities of a given plant and it seemed necessary to conduct further experiments in order to demonstrate conclusively whether this plant can poison or not. So far as the experiments of 1909 only were concerned, it appeared probable that the plant was not poisonous, or if poisonous at all would do harm only under exceptional circumstances.

EXPERIMENTAL FEEDING OF DELPHINIUM MENZIESII TO CATTLE IN 1910.

In 1910, 14 feeding experiments of *Delphinium menziesii* to cattle were carried on with 11 different animals. Of these experiments 9 produced illness and 3 death. The result of these experiments showed that the failure to produce poisoning in 1909 was not due to a lack of toxicity in the plant but to feeding it in too small quantities. Doubtless similar results would have been produced in 1909 had the experiments been continued for a longer time. Table V gives a summary of the feeding experiments with *Delphinium menziesii* to cattle in 1910.

TABLE III.—*Summary of feeding experiments upon cattle with Delphinium barbeyi, 1911.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*						*Pounds.*	
120	600	22	June 8–10	Leaves and stems	Death	Physostigmin, pilocarpin, and strychnin.	Death	36.7	Near station.
115	700	[1]47	June 8–12	do				67.1	Do.
115	700	49	June 20–22	do	Death	Physostigmin, pilocarpin, and strychnin.	Death	70	Do.
92	1,050	85	June 21–23	do	23 hours	Physostigmin, pilocarpin, strychnin, and whisky.	Recovery	81	Do.
631	350	58	June 23–28	do				165.7	Do.
632	400	20.5	July 7–8	do	15 minutes	Physostigmin, pilocarpin, and strychnin.	Recovery	51.2	Kebler Pass.
633	450	21	July 7–11	do	Sick, but not down.		do	46.6	Do.
631	350	15	July 9–11	do				42.9	Do.
635	750	26	July 15	do	13 minutes	Physostigmin, pilocarpin, and strychnin.	Recovery	34.7	Do.
644	650	26	do	do	17 hours	Physostigmin, pilocarpin, strychnin, and magnesium sulphate.	do	40	Do.
634	475	34	July 17–18	do				71.6	Do.
649	500	26	do	do				52	Do.
637	450	23	July 25	Leaves, stems, and flowers	1 hour, 10 minutes on 1st day, at times on 2d day.	Physostigmin, pilocarpin, and strychnin.	Recovery	51.1	Do.
646	500	20	do	do	20¼ hours	Physostigmin, pilocarpin, strychnin, and magnesium sulphate.	do	40	Do.
639	450	41	July 27–28	do	30 minutes	Physostigmin, pilocarpin, and strychnin.	do	91.1	Do.
647	475	38.5	do	do	40 minutes	do	do	81.1	Do.
640	400	36	July 31	do	21 hours	Physostigmin, pilocarpin, strychnin, magnesium sulphate, and whisky.	do	90	Do.
642	550	42	Aug. 1	do	Constipated, not down.		do	76.4	Near station.
636	500	56.5	Aug. 4–5	Leaves, stems, flowers, and seed.				113	Do.
638	525	40	do	Leaves, stems, and flowers				76.2	Kebler Pass.

643	450	42	Aug. 8	Leaves, stems, flowers, and seed.	6½ hours	Physostigmin, pilocarpin, and strychnin.	Recovery	93.3	Near station.
640	400	67	Aug. 8–9	Leaves, stems, and flowers	Constipated, not down.		do	167.5	Kebler Pass.
634	475	129	Aug. 11–13	Leaves, stems, flowers, and seed.				271.4	Near station.
645	650	84	do	do				129.5	Do.
641	450	[2] 11.5	Aug. 16, collected on July 30.	Leaves, stems, and flowers	15½ hours	Physostigmin, pilocarpin, and strychnin.	Recovery	25.6	Kebler Pass.
634	475	[2] 11.5	Aug. 24, collected on July 30.	do	Constipated, not down.		do	24.2	Do.

[1] About.

[2] Dry weight.

TABLE IV.—*Summary of feeding experiments upon cattle with Delphinium menziesii, 1909.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*						*Pounds.*	
112	610	23.4	June 24–28	Leaves, stems, and flowers				38.3	
112	610	4.4	June 29	Leaves, stems, and roots (young plants)				7.2	Kebler Pass.
92	990	2.4	June 26–27	Roots				2.4	
117	620	1.3	July 1	do				2.1	
91	980	5	July 2–3	Leaves, stems, and roots (young plants)				5	Do.
115	610	19.9	do	Leaves, stems, flowers, and seed				32.6	Webber Park.
115	610	61.4	July 10–12	Leaves, stems, flowers, seed, and roots				100.7	Kebler Pass.
112	610	9	July 12	Leaves, stems, and seed				14.7	Webber Park.
91	980	11.8	July 16	Leaves, stems, flowers, and roots				12	Kebler Pass and Webber Park.
97	500	10.6	July 25	Leaves, stems, flowers, and roots				21.2	Near Irwin.

TABLE V.—*Summary of feeding experiments upon cattle with Delphinium menziesii, 1910.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal. weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*						*Pounds.*	
108	600	34.25	May 25–26	Leaves, stems, and roots.				57.1	Near station.
107	600	64.5	May 25–27	Leaves and stems	Fell twice		Recovery	107.5	Do.
107	600	21	May 28	do	About 30 minutes	Physostigmin, pilocarpin, and strychnin.	do	35	Do.
84	750	78	May 30–June 1	Leaves, stems, and flowers.				104	Do.
108	600	64.25	June 3–4	do	Death	Barium chlorid, caffein, and strychnin.	Death	107.1	Do.
117	900	79.5	June 7–9	Leaves, stems, flowers, and seed.	do		do	88.3	Do.
82	1,000	116.5	June 11–14	Leaves, stems, and flowers.	18 hours	Physostigmin, pilocarpin, strychnin, and alcohol.	Recovery	116.5	Near station, north of Salt Lick, and Pass Creek.
118	250	26	June 17–18	Leaves, stems, flowers, and seed.	20 minutes	Physostigmin, pilocarpin, and strychnin.	do	104	Pass Creek.
113	900	56	June 20–22	do	2 hours	do	do	62.2	Do.
609	500	43.75	June 26–27	do	5 minutes		do	87.5	Pass Creek and Webber Park.
609	500	7.5	June 28	Leaves, stems, and flowers.	Sick, but not down		do	15	Kebler Pass.
609	500	18.75	do	Leaves, stems, flowers, and seed.	2 hours and 45 minutes.	Physostigmin, pilocarpin, and strychnin.	do	37.5	Pass Creek.
611	450	43.5	June 29–30	Leaves, stems, and seed.	Death	do	Death	96.7	Do.
614	500	46.5	July 2–3	Leaves, stems, flowers, and seed.	1 hour and 5 minutes		Recovery	93	Do.

A few of the typical cases will be noticed in detail, as follows:

CASE 117.

Case 117 was fed on flowering tops of *Delphinium menziesii* from June 7 to June 9, receiving, all told, 79.5 pounds, or about one-eleventh of its weight. At 9.25 p. m. June 9, when disturbed, he attempted to walk and fell down, recovering himself with little effort. Other than this there were at this time no symptoms of poisoning. He was observed up to 10.30 p. m. and at that time seemed to be fairly well. On the morning of June 10 he was found dead. He lay upon the left side with his head lower than the rest of the body. Some of the contents of the stomach had flowed from the mouth and nostrils. The heart was in diastole, both sides being filled with blood. The pericardial fluid was slightly bloody and abundant. The external walls of the ventricles showed petechiæ. The lungs were congested. The fluids of the pleural and peritoneal cavities were also slightly bloody. The trachea contained some of the contents of the rumen. The inner wall of the trachea was congested and this condition extended into the bronchi. The inner wall of the first stomach was inflamed beneath the mucous membrane, the inflammation being especially deep at the cardiac end of the stomach. The same condition of the wall beneath the mucous membrane was found in the second stomach at the cardiac end. The inner wall of the fourth stomach was also inflamed at the cardiac end. The duodenum was not inflamed near the stomach but there were deep spots of inflammation near the entrance of the bile duct. More or less congestion was found throughout the ileum, this being so deep in some spots as to show through from the outside of the intestine. The left kidney was congested. The brain was congested, probably due in part to the fact that the head was lower than the body. The immediate cause of death was asphyxiation, resulting, partly at least, from the introduction of the contents of the stomach into the trachea, although it seems probable that this was accompanied by respiratory paralysis.

CASE 82.

Case 82 was an old cow weighing about 1,000 pounds. From June 11 to June 14 she ate 116.5 pounds of *Delphinium menziesii* in flower. It was noticed on the morning of June 14 that she was much constipated. She showed no other symptoms of poisoning until 3.20 p. m. of that day, when she was found down. She was able, however, to get upon her feet, but went down again immediately. At 3.26 she was given hypodermically physostigmin salicylate, 1½ grains; pilocarpin hydrochlorid, 3 grains; and strychnin sul-

fate, 1 grain. At 3.28 the respiration was 22. Figures 1, 2, and 3 of Plate XIII show her attitudes at various times between 3.20 and 3.28. She got upon her feet again at 3.28. At 3.30 she trembled, arched her back, and fell, rising again at 3.33. At 3.35 she fell, but was upon her feet again at 3.36. Respiration at 3.43 was 42. There was considerable salivation at this time. At 3.56 she began stepping about uneasily with her head down, and, trembling violently, she staggered and fell. Plate XIII, figure 4, shows her attitude just before she fell, while figure 5 shows her position after she was down, and figure 6 shows her attitude as she was attempting to get up at 3.59. At 4 o'clock her pulse was 112 and rather weak. At 4.01 the pulse was 94. At 4.25 she defecated, probably as the result of the dose of physostigmin salicylate. At this time she showed considerable intestinal discomfort. She continued lying down, but apparently feeling quite comfortable from evening until night. At 5.45 a. m., June 15, she was found in the ditch in the corral with water flowing about her. She was thoroughly chilled and constantly trembling, and there seemed to be little probability that she would live. Apparently she must have risen upon her feet during the night, fallen into the ditch, and was unable to get out. The water was turned off and she was given alcohol in hot water as a drench. Half an hour later she was given a drench of whisky. Soon after this she attempted to get up, and at about 9 o'clock was on her feet. After getting up she urinated copiously. It seemed probable in this case that defecation produced by the physostigmin resulted in relief from the immediate symptoms of larkspur poisoning, and that the alcohol bridged over a period of weakness, due in part to the chill and in part to the effect of the larkspur poisoning. Without the dose of alcohol she would in all probability have died.

Case 113

Case 113 was a steer weighing about 900 pounds. Between June 20 and June 22 he received 56 pounds of *Delphinium menziesii* tops, which included flowers and seeds, the full amount being about one-sixteenth of his weight. At 9.30 p. m. June 22 he was found lying in the corral in a normal manner, but when disturbed he was unable to rise. At 9.35 he attempted to get up, fell over on his side, and was unable to raise himself again. He was given, hypodermically, physostigmin salicylate, 1½ grains; pilocarpin hydrochlorid, 3 grains; and strychnin sulphate, 1 grain. The pulse at this time was 72 and rather weak. Respiration was 16 and fairly deep. While down he was making violent attempts to rise, kicking and lifting his head rather high and then falling back. This action seemed to be more pronounced after the remedy was given, and it was a question

whether it was not partly caused by the peristaltic action resulting from physostigmin salicylate. At 10 p. m. the pulse seemed slightly stronger. He was evidently in pain, as he groaned a great deal of the time. At 11.20 it was found that he had moved himself quite a little distance in the corral and passed a small amount of hard feces. At 11.30 he got upon his feet and walked about the corral. His gait, however, was stiff, the stiffness being particularly noticeable in the hind legs. At 11.44 he passed a considerable amount of feces and acted as though he wished to eat. As he appeared to be very much better at this time, he was left for the night, and was found in good condition at 7 a. m. June 23. He was turned into the pasture at 8.30. In the afternoon of this day he was found in a clump of aspens in the pasture and was driven out. He went about 100 yards in a slow trot, going down a side hill, and fell. This was at 3.55. At 4.05 he began to vomit. His pulse was about 85 and weak. At 4.12 respiration had ceased. The pulse was perceptible for about three minutes, stopping at 4.15. The animal was slightly bloated at first and began bloating rapidly when down. A considerable amount of material from the rumen had been vomited. At the autopsy the heart was found in diastole. The outer walls were slightly inflamed. Both ventricles were dilated and full of blood. The veins under the skin were congested. The nares, larynx, and trachea were full of the material vomited from the stomach, and this material had also extended into the bronchi. The walls of the fourth stomach were greatly inflamed, and the walls of the duodenum, jejunum, ileum, and rectum were slightly inflamed. A microscopic examination was made of the contents of the stomach, and it was found that *Delphinium barbeyi* was present. It seems probable that the animal, after recovering from the poisoning by *Delphinium menziesii* had commenced to eat the *Delphinium barbeyi*, which was fairly abundant in the pasture, and that his death was caused by this dose of the tall larkspur.

CASE 609.

Case 609 was a yearling heifer weighing about 500 pounds, loaned to the station for experimental purposes. Feeding was commenced at 7.05 a. m. on June 26, the material being tops of *Delphinium menziesii*, which at this time was mature and included seeds. On June 26 and 27 she ate 43.75 pounds. The material on June 27 contained flowers as well as seed. Distinct symptoms of poisoning were observed early on the morning of June 28. Before that it had been thought that she was somewhat uneasy, but the symptoms were not positive. At 4.55 a. m. she got up and walked a few steps, trembled, and fell, but at 5 she got upon her feet and after this time was able to stand. She was down only about five minutes. During the

day she ate about 7½ pounds of *Delphinium menziesii*. At 4 p. m. she appeared uneasy. There was occasional forcible expiration and much constipation. After a time her uneasiness seemed to subside and she began to ruminate and appeared hungry. At 5 p. m. she was run around the corral, with no result. Feeding was renewed at 5.15 p. m., and during the evening she received 18.75 pounds of *Delphinium menziesii*, including the seeds. At 9.30 p. m. she was found with her back arched, but appeared fairly well. At 10.15 p. m. she stood with her tail between her legs and her head rather low. The impression was that the poison was taking effect. She started to run about the corral, stumbled and partly fell, but recovered herself, then fell and could not rise. The observer went to the laboratory to get a remedy and on returning found her upon her feet, and she remained upon her feet even after running around the corral. She was left again at about 11.40. During all the time she was watched she was uneasy. She occasionally would expel gas rather violently, and once she moaned. She was evidently very uncomfortable, but not very sick. At 12.10 midnight she was on her feet, but moved around the corral slowly. She began to back uneasily with her head low, and fell and, although making violent efforts to rise, was unable to do so. At 12.15 she was given subcutaneously physostigmin salicylate, 1 grain; pilocarpin hydrochlorid, 2 grains; and strychnin sulphate, 1 grain. She was in great pain, breathed noisily, and occasionally expelled gas from her stomach. She would stretch her legs out rigidly and kick violently, moaning all the time. At 12.40 she passed a little hard feces. At 12.45 her respiration was 40 and continued at about that rate. She perspired copiously and acted like an animal in a violent attack of colic. At 1.25 she raised her head, making efforts to rise, but fell back, striking her head violently upon the ground. This was repeated at 1.30. From this time she seemed to be somewhat easier, although the change was rather gradual. She lay upon her side, breathing noisily. Her legs much of the time were stiff, but the movements were not so convulsive and apparently her pain was less. During the most violent spasms of pain she was given a little ammonia inhaled from saturated cotton. At about 2 a. m. after several violent efforts she succeeded in getting upon her feet, staggered across the corral, but did not fall. She was watched at intervals during the rest of the night and was upon her feet all the time. She was given a little hay and corn meal in the morning and hay at noon. On the following day she appeared to be entirely recovered.

EXPERIMENTAL FEEDING OF DELPHINIUM ROBUSTUM TO CATTLE.

The species of larkspur which has been identified as *Delphinium robustum* and which is quite different from *Delphinium barbeyi* and *Delphinium menziesii* of the Mount Carbon station is abundant in

parts of the Cochetopa and Uncompahgre National Forests. It is more nearly related to the *Delphinium barbeyi* than to *Delphinium menziesii*, and should be classed as one of the tall larkspurs. The entire feeding experiment with this plant was carried on at the ranch of A. J. Hack, of Parlins, Colo. Two animals, Nos. 629 and 630, were used for feeding.

The feeding began at 7.15 a. m. on August 22, 1910. No. 630 ate very little of the larkspur and was not affected by it. No. 629, weighing about 500 pounds, ate on August 22 about 20 pounds, which included leaves, stems, flowers, and seeds. No effect was produced, and at 6 a. m. on August 23 she seemed to be all right with the exception of constipation, but at 10.35 she was found down on her side and unable to rise. She struggled when approached, but was unable to raise herself even upon her belly. At 10.40 respiration was 32 and somewhat irregular. There was some trembling of the muscles of the sides and some salivation. At 10:45 the pulse was 80 and weak. At 11.10 respiration was 50, very irregular and shallow. At 11.34 she arose without any marked difficulty, but at 11.37, after being run about, she went down again, trembling before she fell. With assistance she got upon her feet and started to run, but fell again. She was up again at 11.42 and during the rest of the day seemed to be all right. In the evening she was given more of the *Delphinium robustum*, it being estimated that she ate about 8 pounds. On the morning of August 24 she was found down and unable to rise. A little later she arose with some difficulty, but fell, getting upon her feet again at 6.35, when she immediately fell and was unable to rise. At 6.40 she got up and walked away. She started to run and fell, but immediately got upon her feet, only to fall again, trembling as she fell. At 6.45 she got upon her feet and walked about in a normal manner. She was seen frequently during the forenoon and seemed to be all right, with the exception of some constipation.

It will be noticed that the symptoms as recorded are exactly comparable with those found in the cases of poisoning by *Delphinium barbeyi* and *Delphinium menziesii*.

EXPERIMENTAL FEEDING OF DELPHINIUM CUCULLATUM TO CATTLE.

During the summer of 1912, at the Greycliff station, *Delphinium cucullatum* was fed experimentally to six head of cattle with resulting symptoms of poisoning in four, none of the cases resulting fatally. One was only slightly sick and received no remedy. The second was treated with arecoline with no apparent good results, but recovered after treatment with magnesium sulphate, a glycerin enema, and a hypodermic injection of whisky. The others were treated in the routine way worked out at Mount Carbon with physostigmin and pilocarpin and recovered. The symptoms were strictly comparable with those produced by the other species of Delphinium and it does not

seem necessary to give the history of the cases in detail. In the discussion later in this paper the minor points of difference will be brought out. Table VI gives the summary of these feeding experiments.

TABLE VI.—*Summary of feeding experiments upon cattle with Delphinium cucullatum.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.
	Pounds.	*Pounds.*	1912.	
650	550±	12.5	June 28–29	Leaves and stems.
653	700±	18.5	June 30–July 1	Do.
654	600±	21	July 23	Leaves, stems, and flowers.
652	500±	24.5	August 8–9	Leaves, stems, flowers, and seeds.
653	700±	2.5	August 30–31	Leaves, stems, and seed.
651	550±	17.5	September 3	Do.

No. of animal.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal weight.	Location from which plant fed was obtained.
				Pounds.	
650	Slightly sick; not down		Recovery	22.7	Cabin corral.
653	18 hours, 40 minutes	Arecolin, strychnin, magnesium sulphate, glycerin, and whisky.	do	26.4	Do.
654	4 hours, 15 minutes, first attack; 20 hours, 30 minutes, second attack.	Physostigmin, pilocarpin, strychnin, linseed oil.	do	35	Do.
652	25 minutes, first attack; 14 hours, second attack.	Physostigmin, pilocarpin, and strychnin.	do	49	Do.
653				3.5	Do.
651				31.8	Do.

POISONING OF HORSES BY LARKSPUR.

Apparently there are no accounts of the poisoning of horses by larkspur. Among the stockmen it is a general belief that horses are not poisoned by this plant and can be grazed with impunity in localities where cattle are certain to suffer from the poison. In some localities in the Sierras where many cattle have been lost within limited areas, the ground has been fenced in and successfully used for pasturing horses. Although the belief is general that horses are not poisoned by larkspur, it does not follow that they can not be. Accordingly the following experiments were undertaken with cases 72 and 78.

CASE 72.

Case 72 was a horse about 4 years old which had been used in previous feeding experiments. During July 9 and 10 he ate 11 pounds of leaves and stems of *Delphinium barbeyi* without any effect. Another experiment was made, commencing on the morning of August 24 and continuing until September 4. During this time he ate 192 pounds, or, on the basis of 1,000 pounds of weight, 274.3 pounds. The feeding was then interrupted on account of storms,

but was resumed on September 9. From this time until September 14 he ate 78.25 pounds of *Delphinium barbeyi*. The material fed in these later experiments was mature and dry. No effect resulted from the larkspur feeding except that part of the time the horse seemed sleepy and lifeless. It should be noted that this feeding was rather late in the season, when, as shown elsewhere, the larkspur is only slightly toxic.

CASE 78.

Case 78 was a horse weighing about 600 pounds, which had already been used at Hugo in the loco experiments. An attempt was made early in July to feed it both *Delphinium barbeyi* and *Delphinium menziesii*, but without any effect. On August 23 it was brought into the corrals in order to try a prolonged feeding experiment with *Delphinium barbeyi*. The material given was collected in Kebler Pass and consisted of tops, including the fruit. The animal was fed from August 24 to September 2, inclusive. During this time it ate 168½ pounds, or, in the ratio of its weight, the quantity eaten was as 1 to 3.6. No effect of the feeding was noticed until September 2. Between 10 and 11 o'clock of the morning of September 2 it was noticed that the action of the hind legs was stiff and that the animal acted as if he did not have complete control of his legs. There was some trembling of the muscles of the flanks and twitching of the muscles of the lips and nostrils. The abdominal muscles contracted as though in pain. In walking he straddled with his hind legs and appeared weak behind. He was constantly moving about, apparently from pain. The back was arched up, and he was very much constipated. At 11.25 he was given some hay and commenced to eat it, but while eating stepped about uneasily as though in pain. At 12.15 he was found down, but was started up and got upon his feet without any difficulty, although his movements after rising were somewhat uncertain. After rising he kept walking about, evidently feeling very uncomfortable. He lay down again at 12.25. His respiration at this time was 78. Figures 1, 2, 3, and 4 of Plate XIV show various attitudes assumed during his illness; figure 1 shows clearly the discomfort under which the animal was laboring; figure 2 shows him after he lay down; figure 3 shows his attitude at 1.08, when he was most severely ill; and figure 4 shows him a little later than this when he was upon his feet but still feeling great discomfort. At 1.55 he was lying down again, and when started and run around the corral he moved readily, showing little tendency to stagger or to fall. At 3 p. m. he was found standing in the corral, his lips no longer trembling, and he no longer had a tendency to walk about uneasily as earlier in the day. His gait was slow, however, and he was sleepy. At this

time evidence of pain was less marked. No further pronounced symptoms appeared during the day of September 2.

On the morning of September 3 he still exhibited some trembling of the muscles of the hind legs and of the flanks and his gait showed the same symptoms of stiffness as seen on the preceding day, but during the day his condition improved. On this day he ate 6 pounds of leaves, stems, and seeds. On the morning of September 4 he appeared to be in good condition. The feeding was resumed, and he ate about 12 pounds of *Delphinium barbeyi*. During the latter part of the forenoon and in the afternoon he again showed distinct symptoms of poisoning. The back was arched much of the time and he straddled in walking. There was distinct evidence of abdominal pain. During much of the time he exhibited trembling in the superficial muscles. Sometimes in lying down he would groan, evidently being in severe pain. Gradually, however, he recovered, and on the morning of September 5 appeared to be again in normal condition.

He was fed again, from September 9 to September 14, receiving fresh material of *Delphinium barbeyi* collected at Kebler Pass. During this time he ate 126.75 pounds. There were no distinct evidences of poisoning from this feeding, although he appeared somewhat dull.

The results of these experiments seemed to prove conclusively that horses can be poisoned with larkspur and that they have the same general symptoms as cattle. Table VII gives the summary of these feeding experiments.

TABLE VII.—*Summary of feeding experiments upon horses with Delphinium barbeyi, 1909.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.
	Pounds.	*Pounds.*	1912.	
72	700	11	July 9–10	Leaves and stems.
78	600	168.5	Aug. 24–Sept. 2	Leaves, stems, and seed.
72	700	192	Aug. 24–Sept. 4	Do.
78	600	6	Sept. 3	Do.
78	600	12	Sept. 4	Do.
72	700	78.25	Sept. 9–14	Seed and seed stems.
78	600	126.75	do	Leaves and stems.

No. of animal.	Time sick until able to stand.	Remedy used.	Result.	Amount fed to 1,000 pounds of animal weight.	Location from which plant fed was obtained.
				Pounds.	
72				15.7	Near station.
78	Sick, but able to stand.		Recovery	280.8	Kebler Pass mostly.
72				274.3	Do.
78	Sick, but able to stand.		Recovery	10	Kebler Pass.
78	do		do	20	Do.
72				111.8	Do.
78				211.2	Do.

For our purpose it did not seem necessary to carry on any further experimentation with horses as there is no reason to think that they are ever poisoned upon the range. Observation of horses on the range and in pastures containing larkspurs showed that these animals do not eat the larkspur early in the season. In the fall after the tall larkspur has become dry, horses, like cattle, seem to have a fondness for the larkspur leaves, although they do not eat them so greedily as do the cattle. Inasmuch as the larkspur at this time is not poisonous, no harm results from this feeding.

EXPERIMENTAL FEEDING OF DELPHINIUM BARBEYI TO SHEEP IN 1910.

Five experiments were carried on of feeding *Delphinium barbeyi* to sheep. These experiments were commenced June 3 and continued until July 17. Three of the animals, Nos. 118, 104, and 114, were fed tops of the plant, including the leaves and stems. The other two, Nos. 108 and 116, were fed tops, including the leaves, stems, and flowers. Table VIII gives a summary of these experimental feedings.

Case 118, weighing 97 pounds, was fed 67.75 pounds between June 3 and July 22 without any results. Case 104, weighing 90 pounds, was fed 68 pounds between June 23 and July 5 without any injurious results. Case 114, weighing 65 pounds, received 31.75 pounds between June 23 and July 5 without results. Case 108, weighing 94 pounds, was fed 104 pounds between July 6 and July 17, or 10 pounds more than its own weight, without being poisoned. Case 116, weighing 93 pounds, received 121 pounds between July 6 and July 17, or 28 pounds more than its own weight, without being affected.

Thus of these 5 sheep, eating from 48.8 to 130.1 pounds, on a basis of 100 pounds average weight, none were injuriously affected by the plant.

EXPERIMENTAL FEEDING OF DELPHINIUM BARBEYI TO SHEEP IN 1911.

In 1911 two experiments were made of feeding *Delphinium barbeyi* to sheep. Although the general results of the work of 1910 were conclusive, it seemed best to feed some of the plant at the early stages of its growth in order to make sure that it was not poisonous at that time.

Sheep 134, weighing 140 pounds, was fed from June 19 to June 25, 49 pounds of the leaves and stems of *Delphinium barbeyi* before blossoming. This was at the rate of 35 pounds per 100 pounds of weight of the animal.

Sheep 135, weighing 136 pounds, between the same dates, was fed 37 pounds of the same material, or 27.2 pounds per hundredweight of the animal.

Neither of these sheep showed any effects from the feeding, and inasmuch as the amount fed, relative to the weight of the animal, was

so much larger than that necessary to poison cattle, it was deemed conclusive evidence that the plant at this stage is not poisonous to sheep.

TABLE VIII.—*Summary of feeding experiments upon sheep with Delphinium barbeyi, 1910 and 1911.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Amount fed to 100 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*	1910.		*Pounds.*	
118	97	67.75	June 3–22	Leaves and stems	69.9	Near station.
104	90	68	June 23–July 5	do	75.5	Do.
114	65	31.75	do	do	48.8	Do.
108	94	104	July 6–17	Leaves, stems, and flowers	110.6	Do.
116	93	121	do	do	130.1	Do.
			1911.			
134	140	49	June 17–25	Leaves and stems	35	Do.
135	136	37	do	do	27.2	Do.

EXPERIMENTAL FEEDING OF DELPHINIUM MENZIESII TO SHEEP IN 1910.

Four sheep weighing approximately 100 pounds each were fed various quantities of *Delphinium menziesii*. Table IX gives a summary of these experimental feedings.

Sheep 113 was fed 32.75 pounds of roots, the feeding continuing from June 2 to June 13. On June 13 the available supply of roots for feeding was exhausted and the sheep was given the tops, including leaves and flowers. This feeding was continued through June 22, the animal having received, altogether, 50.25 pounds of this material. Sheep 125 was fed 111.75 pounds of tops, including leaves, stems, and flowers, the feeding continuing from June 2 to June 16. Sheep 119 was fed from June 15 to June 26, the material being the entire top, including leaves, stems, flowers, and seeds. During this time the animal ate 101 pounds, or very nearly its own weight. During the same period, June 15 to June 26, sheep 123 was fed 73.75 pounds of the same material.

The *Delphinium menziesii* fed to sheep 113 during the first experiments of root feeding was collected near the camp. All the rest of the material fed to the sheep was collected at Pass Creek Park and was of fairly mature plants. The feeding of this plant to sheep produced no injurious effect whatever. The animals did not even lose much in weight, and that little could be accounted for by reason of confinement and the fact that they were being fed but a single variety of plant.

It should be added that sheep 160 ate in one day, on the basis of 100 pounds of weight, 5.98 pounds, and sheep 177, 6.9 pounds. In the experiments of 1910 and 1911 sheep 118 ate in one day 6.7 pounds; sheep 114, 6.5 pounds; sheep 135, 7.4 pounds; sheep 134, 7.8 pounds;

FIG. 1.—CASE 78, SHOWING DISCOMFORT.

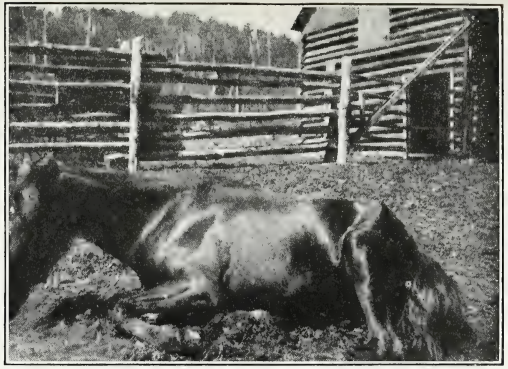

FIG. 2.—CASE 78, LYING DOWN BUT ABLE TO HOLD HIS HEAD ERECT.

FIG. 3.—CASE 78, WHEN FEELING THE WORST.

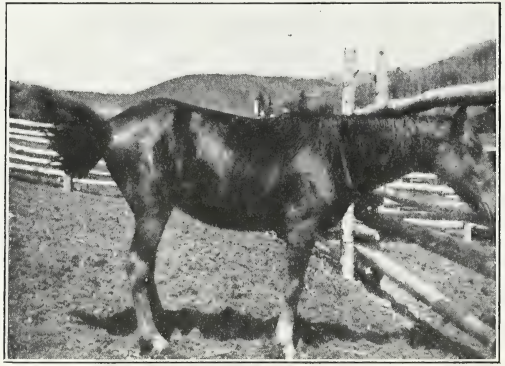

FIG. 4.—CASE 78, A LITTLE LATER THAN FIG. 3, WHEN ABLE TO STAND.

FIG. 1.—SHEEP FEEDING UPON DELPHINIUM MENZIESII.

FIG. 2.—ANOTHER VIEW OF SHEEP FEEDING UPON DELPHINIUM MENZIESII.

sheep 108, 11.7 pounds, and sheep 116, 15.6 pounds. Inasmuch as the toxic dose for cattle, as is shown later, is from 3 per cent and upward of the animal's weight, the sheep ate, relatively to their weight, from 2 to 5 times as much as is necessary to poison cattle without harmful results.

TABLE IX.—*Summary of feeding experiments upon sheep with Delphinium menziesii, 1910.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Amount fed to 100 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*			*Pounds.*	
125	100	111.75	June 2–16	Leaves, stems, and flowers	111.75	Pass Creek.
113	100	32.75	June 2–13	Roots	32.75	Near station
113	100	50.25	June 13–22	Leaves, stems, and flowers	50.25	Do.
119	106	101	June 15–26	Leaves, stems, flowers, and seed.	95.3	Pass Creek.
123	90	73.75	do	Leaves, stems, and flowers	81.9	Do.

EXPERIMENTAL HERDING OF SHEEP UPON DELPHINIUM MENZIESII.

Delphinium menziesii was particularly abundant in Pass Creek Park, near the Mount Carbon Station. When the plant was in blossom the whole park seemed to be colored purple. Plate XV, figures 1 and 2, show sheep feeding and give a good idea of the abundance of the plant in the park. It was thought desirable to try an experiment of close herding a bunch of sheep upon the larkspur. If *Delphinium menziesii* would poison sheep upon the range, symptoms ought to be developed by such close herding, for if they ate freely it would be evident that a much larger quantity would be eaten than under any ordinary circumstances that would prevail in range feeding. Accordingly, on June 14, 19 sheep were taken from the station to Pass Creek Park and were kept until the afternoon of June 17. During the day they were herded upon the larkspur area, and corralled at night in an old cabin. Most of them ate very freely of the *Delphinium menziesii*. Notes taken in regard to the individual sheep show that five may be considered to have eaten only a little. All the rest, however, ate a considerable quantity. They were not allowed to stray from the larkspur patch, and the feeding while they were herded resulted in clearing swaths running through the larkspur area where most of the plants had been eaten. During this time they ate very freely, apparently relishing the taste of the larkspur. Plate XV, figures 1 and 2, which were snapshots taken during the feeding, show how readily they took to the plant. The sheep were watched very closely for possible symptoms of larkspur poisoning. No such symptoms, however, developed. All were brought back to the camp on June 17 not only none the worse for their ex-

perience but apparently, if anything, benefited by the opportunity of free pasturing. During the summer the band of sheep was corralled nights in a small pasture which during the month of June was almost entirely covered with *Delphinium menziesii*. All of this plant was eaten out with the other plants growing in the pasture and no injurious effect was produced upon any of the sheep.

These experiments, in conjunction with the corral feeding experiments, seemed to the station force to prove conclusively that sheep can eat *Delphinium barbeyi* and *Delphinium menziesii* with impunity, and that no fear of poisoning need be entertained from pasturing sheep on a range containing these plants.

EXPERIMENTAL FEEDING OF DELPHINIUM ANDERSONII TO SHEEP IN 1911.

Inasmuch as the belief is very common among the sheepmen of California and Oregon that sheep are poisoned by eating the roots of *Delphinium andersonii*, two experiments were made of feeding the roots of this plant. The material was collected at McDowell's Creek, near Lakeview, Oreg., on June 24, 1911, and forwarded to the station at Mount Carbon. As it is somewhat difficult to make sheep eat the roots of the plant, and as the quantity was limited, it was decided to administer the material in the form of a drench. The roots after being washed were ground fine and mixed with enough water to permit of their administration. Two animals were used.

Sheep 155, weighing 131 pounds, was brought in for experiment on August 9. At 11.30 a. m. on August 11 it was given 200 grams of dried roots and on August 12 at 11 a. m. and 2.30 p. m. it was given the same amount. It thus received 1.3 pounds of the roots. Inasmuch as these had been air dried it would be equivalent to at least twice that amount of fresh roots.

Sheep 136, weighing 153 pounds, was brought in for feeding on August 13. At 10.30 a. m. on August 14 it was given 200 grams of the roots. The same quantity was administered at 2.20 p. m., 4.45 p. m., and 7.45 p. m., and 150 grams were given at 9.45 p. m. Thus, this animal received between 10.30 a. m. and 9.45 p. m. 2.1 pounds of dried roots. This would be equivalent to at least 4.2 pounds of fresh material. It is highly improbable that the sheep upon the range, grazing upon larkspur, would obtain anything like this amount of larkspur roots, so that this experiment in conjunction with the experimental work of feeding roots of other larkspurs at Mount Carbon seems to be fairly conclusive that sheep are not poisoned by eating roots of *Delphinium andersonii*. Table X gives the results of this experimental feeding.

TABLE X.—*Summary of feeding experiments upon sheep with Delphinium andersonii, 1911.*

No. of animal.	Weight of animal—		Amount of plant fed.	Date of feeding.	Part of plant fed.	Amount fed to 100 pounds of animal weight.	Location from which plant fed was obtained.
	Before.	After.					
	Pounds.	*Pounds.*	*Pounds.*	1911.		*Pounds.*	
155	131	126	[1] 1.3	Aug. 11–12	Roots..	1	McDowells Creek, Oreg.
136	153	149	[1] 2.1	Aug. 14	...do...	1.4	Do.

[1] Dry weight.

EXPERIMENTAL FEEDING OF DELPHINIUM BICOLOR TO SHEEP IN 1912.

The preceding experiments of feeding *Delphinium barbeyi*, *D. menziesii*, and *D. andersonii* with negative results rendered it extremely probable that none of the species of larkspurs are poisonous to sheep. It was assumed that probably *Delphinium bicolor*, the Montana low larkspur, would be harmless. In the summer of 1912 two sheep were fed upon this plant, Table XI, giving the details. No symptoms of poisoning occurred in either case, although they received much more than it is at all probable they could obtain when grazing.

TABLE XI.—*Summary of feeding experiments upon sheep with Delphinium bicolor, 1912.*

No. of animal.	Weight of animal.	Amount of plant fed.	Date of feeding.	Part of plant fed.	Amount fed to 100 pounds of animal weight.	Location from which plant fed was obtained.
	Pounds.	*Pounds.*			*Pounds.*	
160	92	23.5	June 23–July 2	Leaves, stems, fruit, and some flowers.	25.54	Geo. Hughes's.
177	51–48	16.5	July 5–12.....	Leaves, stems, and fruit....	32.35	Do.

PART III.—RESULTS AND CONCLUSIONS.

ANIMALS AFFECTED BY LARKSPUR POISONING.

Cattle.—The experimental work resulted in the confirmation of the general opinion of the poisonous effect of the larkspurs upon cattle.

Horses.—Horses may be poisoned by larkspur, but they do not voluntarily eat enough of the plants to harm them. They eat more or less of it when grazing, but there is no evidence that they are ever poisoned by it under ordinary range conditions.

Sheep.—As the result of the feeding experiments with *Delphinium barbeyi* and *Delphinium menziesii* at the Mount Carbon station, the definite conclusion was reached that these two plants do not have

any poisonous effect upon sheep. Not only were no poisonous effects produced by close feeding upon the plant but the animals did not lose weight and seemed to thrive upon larkspur as a fodder. Inquiry among the stockmen of the Gunnison and neighboring stock ranges brought out the fact that there is a general belief among them that larkspur is never poisonous to sheep. Sheep have been grazed upon the range not many miles from the Mount Carbon station for many years and there are no records of losses from larkspur poisoning. Inasmuch as the feeding of *Delphinium andersonii* and *Delphinium bicolor* was also without result it seems probable that all species of larkspur are harmless so far as sheep are concerned. These results are in harmony with those reached by S. B. Nelson, in Washington, but apparently distinctly contradict the work of Wilcox, in Montana (1897).

A careful examination of Wilcox's original paper shows that the evidence in regard to larkspur poisoning in Montana is hardly conclusive. He finds that a certain number of sheep died and that these animals had been eating larkspur, but it does not follow, of course, that larkspur was the cause of the fatal results, and, with the exception of giving extracts to three lambs, no experimental evidence of larkspur poisoning is adduced. It may be considered possible that the symptoms noted from the extracts might be explained in other ways. It should be noted, however, that the detailed symptoms of larkspur poisoning of sheep, as given by Dr. Wilcox, correspond very closely with the symptoms as given by other authors and with those noted at the Mount Carbon station.

A visit was made to the locality in Montana where this sheep poisoning had taken place, and conversation with the owners of the sheep showed that not only were they very skeptical in regard to the alleged fact that larkspur is the cause of the death of the sheep, as described by Dr. Wilcox, but also that they and other sheepmen of the neighborhood did not consider the larkspurs poisonous to sheep. The results of the work at Mount Carbon and at Greycliff seem to indicate that, in all probability, larkspurs need not be feared by sheep owners. In California and Oregon there is among the sheepmen a belief, widespread and persistently adhered to, that many sheep are lost in the spring from eating larkspur roots. This belief applies, apparently, to *Delphinium andersonii*. This species has a stout stem and grows in a loose soil, so that grazing animals can pull up the roots. It seemed possible that sheep might be poisoned in this way in California and Oregon, even if they were not harmed in Colorado. The experimental feeding of the roots of *Delphinium andersonii* (p. 58), taken with the other results of feeding sheep, makes it probable that the sheepmen are mistaken in

their idea that the roots of *Delphinium andersonii* are poisonous to sheep.

The somewhat suprising result of the feeding work upon larkspur, showing that of two animals so similar in their physical organization as cattle and sheep one is poisoned and the other not affected has, of course, some physiological explanation. Just what this is has not been determined experimentally. It has been shown, however, that sheep excrete the alkaloid in their urine, and it may be, perhaps, assumed that they excrete with sufficient rapidity to remove the poisonous principle before toxic symptoms appear. It should be noted in this connection that there is still a possibility that the alkaloid might be given experimentally in a quantity so great that the excreting powers of the sheep would be unable to remove it in time to prevent intoxication. It is intended later to complete this experimental study. The experiments do show conclusively, however, that quantities, relatively to the size of the animals, several times as great as those necessary to poison cattle do not affect sheep, and that sheep on the range are for all practical purposes immune to larkspur poisoning.

If it is true, as we think it is, that sheep can feed upon the larkspur, not only with impunity, but with actual benefit to themselves, it would appear possible that on ranges where heavy losses of cattle have taken place because of larkspur poisoning sheep can graze with no loss. The question may be raised whether certain ranges could not be profitably changed from cattle ranges to sheep ranges on this account or whether it might not be possible, inasmuch as the losses of cattle from larkspur poisoning are largely confined to the earlier part of the season, to graze sheep upon the range during the early part of June or until they had eaten off the low larkspur and then admit cattle.

RECORDED SYMPTOMS OF LARKSPUR POISONING.

Hahn, in his general article on Delphinium in the Dictionnaire Encyclopédique des Sciences Médicales, quotes Orfila. He states that the symptoms of poisoning by Delphinium are nausea, vertigo, weakness, and convulsions, followed by death. Falck and Rörig, 1852, state the symptoms as nausea, salivation, restlessness, convulsions, and death produced by asphyxia and paralysis of the heart. The symptoms as quoted by these two authors may be considered as typical of those reported by investigators of the European Delphiniums.

Macgregor, in 1908, in telling of the symptoms of poisoning in a horse says that it became dull, its pulse was weak, and there was excessive salivation and deglutition, with attempts at vomiting.

Knowles, in 1897, in detailing the symptoms, says that the animals stray about, become dull, and when started go on a straight line until an obstacle is met, then fall. They rarely bloat. There is a dribbling of saliva and a champing of the jaws. Wilcox, in 1897, states that the symptoms of larkspur poisoning resemble those of aconite poisoning. The first signs are a general stiffness and a straddling, noted especially in the hind legs. The stiffness becomes more pronounced until walking is very difficult and evidently painful. Soon there are manifested involuntary twitchings of the muscles of the legs and sides of the body. There is a loss of control and coordination of the muscles. Ordinarily there is no increase in the quantity of the saliva, no champing of the jaws or attempts at swallowing. At first the pulse is less frequent and the respiratory movements are lessened, while the temperature is lowered. Toward the last the respiration is very rapid. The air in the lungs is not renewed and the animal dies of asphyxia or suffocation. In the latter cases the involuntary movements become more frequent and more severe. All four legs tremble and shake violently. The muscles of the body contract spasmodically until the animal totters over and dies in violent spasms.

In Chesnut and Wilcox, 1901, the symptoms are stated practically like those already detailed by Wilcox. They say that the animal generally falls and gets on its feet a number of times, while the muscles of the sides and legs quiver spasmodically. This quivering of the muscles is considered a very characteristic symptom. There is a slight increase in the quantity of saliva and the animal dies in violent convulsions. The symptoms of poisoning from the low and the tall larkspurs are practically the same.

In comparing the symptoms as detailed by these authors it is noticed that there is a good measure of general agreement, and we can say that the characteristic symptoms of Delphinium poisoning are nausea, weakness, excessive salivation, twitching of the muscles of the sides and legs, and convulsions.

It may be added that the reports of the symptoms of larkspur poisoning as given by stockmen all through the region where larkspur is abundant agree very well with those detailed above by these authors. It is said by many of the stockmen that when a poisoned animal is started suddenly it runs a short distance, then falls; it may pick itself up and run a little farther, but eventually it falls and dies. Some of them state that poisoned animals froth at the mouth, and most of them agree that the animals die in spasms.

SYMPTOMS OF LARKSPUR POISONING OBSERVED IN THE EXPERIMENTAL WORK.

In the animals fed experimentally in the corrals the first indication of the poisonous effect of larkspur was that they no longer cared to

eat, and became uneasy, stepping about as though uncomfortable. As the animal walks about the corral the gait becomes "stiff" and the hind legs are ordinarily spread somewhat widely apart, as though it were bracing itself against falling. It walks uncertainly, staggering more or less. If the poison is sufficient in quantity, after moving a short distance the animal falls. In falling it ordinarily goes down very suddenly, the legs sometimes appearing to crumple up. The forelegs give out first, and the animal goes down, frequently with the head extended and the chin lying upon the ground; then goes completely down. In the less acute cases the animal goes down and lies with the head erect. If the case is acute, it will fall over upon its side, lying flat upon the ground, sometimes moving the head up and down.

If frightened in this position, the animal may kick violently. Usually it is impossible for it to get upon its feet again immediately after falling, and after making two or three more or less violent attempts it gives up absolutely. In a short time it will usually get up and may move about. Soon it commences to step about uneasily, ordinarily backing, the back arches up, the head is held low, it trembles, and, after one or more attempts to save itself from falling, goes down as before. This may be repeated a considerable number of times. The pictures show quite well the attitudes assumed by the animals under these circumstances.

When the poisoning has a fatal result the animal may lie for some time with labored breathing before it dies. If it recovers, as the effect of the poison passes off it stands upon its feet longer each time after falling, and eventually walks off, very much as if nothing were the matter. In cases of mild poisoning it sometimes happens that the animal falls, and when it gets upon its feet walks off apparently perfectly well. If under such circumstances it is hurried, it will go down again, with the same symptoms as before.

On the range commonly the first symptom noted is the falling of the animal; it goes down suddenly and generally is unable to rise immediately. Sometimes, if cattle which are apparently all right are driven hurriedly for a few minutes, individuals will fall. The same thing was noticed in the experimental animals; some that had shown no preceding symptoms would suddenly fall after being run about the corral.

The symptoms of poisoning from *Delphinium barbeyi*, *D. menziessi*, *D. robustum*, *D. bicolor*, *and D. cucullatum* were so nearly identical that they could not be distinguished. The time of complete prostration, by which is meant the time during which an animal is unable to continue standing upon its feet, varies in accordance with the acuteness of the attack. In the cases in 1909, which were all of

Delphinium barbeyi poisoning, the average time of the animals experimented upon was 3 hours and 25 minutes; the shortest time was a half hour, and the longest 13 hours. Of the animals poisoned by *Delphinium barbeyi* in 1910 the shortest was 16 minutes and the longest 15 hours and 16 minutes. The average of the 17 cases observed was 2 hours and 7 minutes. In 1911 there were 11 cases of animals made sick by *Delphinium barbeyi*. Of these the shortest period was 13 minutes and the longest 23 hours, with an average of 9 hours and 38 minutes.

Of 6 cases of *Delphinium menziesii* in 1910 the shortest period was 5 minutes and the longest period 2 hours and 45 minutes, with an average of 1 hour and 7 minutes.

In the single case of *Delphinium robustum* which was observed in the Cochetopa Forest, the animal was down during its first attack for 1 hour and 7 minutes, and during the second attack on the succeeding day it was down 40 minutes.

In the case of cattle poisoned by *Delphinium cucullatum* at Greycliff, one was not down at all, and, of the others, one was down 18 hours and 40 minutes, while each of the remaining two had two attacks, the second in both cases being very prolonged. No. 654 was down in the second attack 20 hours and 30 minutes.

In almost all cases the evidence was clear that the animals were nauseated. They frequently moved the head back and forth, sometimes shaking it from side to side, these movements clearly indicating a condition of nausea. As the sick animals lay upon the ground, there was often belching of gas at frequent intervals, caused by this condition of nausea. In the cases where vomiting actually took place, the animals were almost sure to die. Of all the experimental animals observed at Mount Carbon, only one that vomited survived. In all the animals that vomited and died, more or less of the contents of the rumen were found in the trachea and bronchial tubes.

The movements of the head also indicated in most cases more or less abdominal pain. Frequently this pain was evidently very severe. The animals were always constipated, sometimes severely so, and without doubt this constipation was connected with the abdominal pain.

Temperatures were taken in a considerable number of cases, both in 1909 and in 1910. These temperatures varied from 101.2° to 102.6° F. There is evidence from this that temperatures, so far as observed, were practically normal. It has been stated by some authors that the temperature at the beginning of the attack is lower. From the observations of the Mount Carbon experimental animals there was no reason to think that larkspur poisoning caused any change whatever in the temperature.

The rate of respiration was noted in a large number of the cases in both years. In general it ran very high. The highest noted was 123, in the case of No. 604, a yearling heifer. Generally speaking, however, it did not go above 60 to 70. In the case of No. 604, the respiration was noted at various periods between 3.15 and 4.22 p. m., the rates observed being 100, 123, 103, 58, 60. In the case of No. 610, in 1910, between 11.45 a. m. and 6.40 p. m., the numbers indicating the rapidity of respiration were 80, 60, 60, 85, 44, 40, 28, 24. These two cases may be considered as typical of the general course of respiration in cases of poisoning. Generally speaking, the respiration was highest and shallow at the most acute stage of the attack and gradually diminished and became deeper as the effects of the poisoning passed off. In nearly all cases, however, even if the animal had apparently entirely recovered, the rate of respiration was still quite high.

The pulse also was noted in a considerable number of cases, and this, as would be expected, was also rapid. The highest observed was 150 in the case of No. 618. Generally speaking, in the acute cases, the pulse ran well toward 100 and was very weak and, as the effect of the poison passed off, would progressively become slower and stronger. In some few cases the pulse during the stage of poisoning was rather low, as, for example, in case of No. 113 in 1909, where the pulse was 50. It immediately, however, went up to 74.

Salivation was not present in all cases, but it was noted in a number of the sick animals. Of the 22 cases sick at the station from eating *Delphinium barbeyi* in 1910, 9 showed more or less marked salivation. It was not a universal symptom but was a common one. Of course, the administration of the remedy physostigmin and pilocarpin increased the salivation, but this symptom was noted before the administration of the remedy, and in cases where no remedy was given.

It is stated by some authors that in larkspur poisoning there is a loss of control of the muscles and that the animals die in violent spasms. This was hardly true of the experimental animals at Mount Carbon. There were involuntary contractions of many of the muscles of the body. These contractions were particularly pronounced in some cases in the muscles around the mouth and nose, which contracted so as to produce a condition of continuous movement of the muzzle. In one or two cases this movement extended to the mandible. The muscles of the shoulders, flanks, and hips contracted spasmodically, and sometimes there appeared to be a muscular trembling over the whole surface of the body. This trembling was much more marked when the animals were standing than when they were down. When down, some of the animals kicked about to some extent, but there

did not appear to be a lack of coordination, and the movements of the animals, while perhaps they might be described as convulsive, could hardly be considered as the movements of violent spasms or convulsions. When the animals attempted to rise, the difficulty, apparently, was weakness rather than a lack of coordination of the muscles, and the kicking of the animals appeared to be due to voluntary attempts to rise rather than to involuntary and spasmodic contractions of the muscles of the legs. It did not seem to the observers that the animals could be said to have convulsions or spasms.

Bloating occurred in some of the cases, but was not a common symptom. In the cases where it was noticed, it seemed to come as one of the later results of the poison. The bloating doubtless adds much discomfort to the animal, and if it lies with the head lower than the rest of the body, may cause death. It is a matter of common knowledge that when animals die of larkspur poisoning upon the range they bloat very quickly, and it seems probable that death may in some cases be immediately caused by the mechanical effects of the bloating.

Recovery from larkspur poisoning is ordinarily very rapid. The animal, after becoming well enough to rise, soon walks away, in a short time begins to eat, and after two or three days shows no effects of the poisoning. Some stockmen believe that cattle do not thrive after being poisoned by larkspur, but from the experimental work it appeared that no permanent injury was caused. Several of the animals were fed upon the larkspur repeatedly in the same season with no bad results in their condition, except the loss of flesh during the days when the experiments were being carried on. In these experiments of using animals repeatedly they were poisoned as readily the second and third times as the first, or, in other words, there is no evidence from the experimental work of acquired toleration; on the other hand, they were no more susceptible to the effects of the poison because of the repeated feedings.

THE TOXIC DOSE OF LARKSPUR.

It was important for practical purposes to determine how much larkspur was necessary to produce poisonous effects. The work of the first season alone did not give very definite indications of the quantity of larkspur necessary to produce poisoning, but taken in conjunction with the work of the succeeding seasons, seems to give results that are quite exact.

From the accompanying charts (see figs. 6 to 12) one can see the toxic dose of larkspur, this being reduced to a uniform scale for animals weighing 1,000 pounds. They show the quantities of larkspur necessary to produce the poisoning, the dates of the experiments, and the length of time during which the plant was fed. The

figures indicate the number of the animal in each case. The letter *S* indicates that the animal was fed seeds, and the letter *L* that leaves were used.

At first glance these charts do not seem to be very instructive. It will be seen that the quantities of *Delphinium barbeyi* necessary to

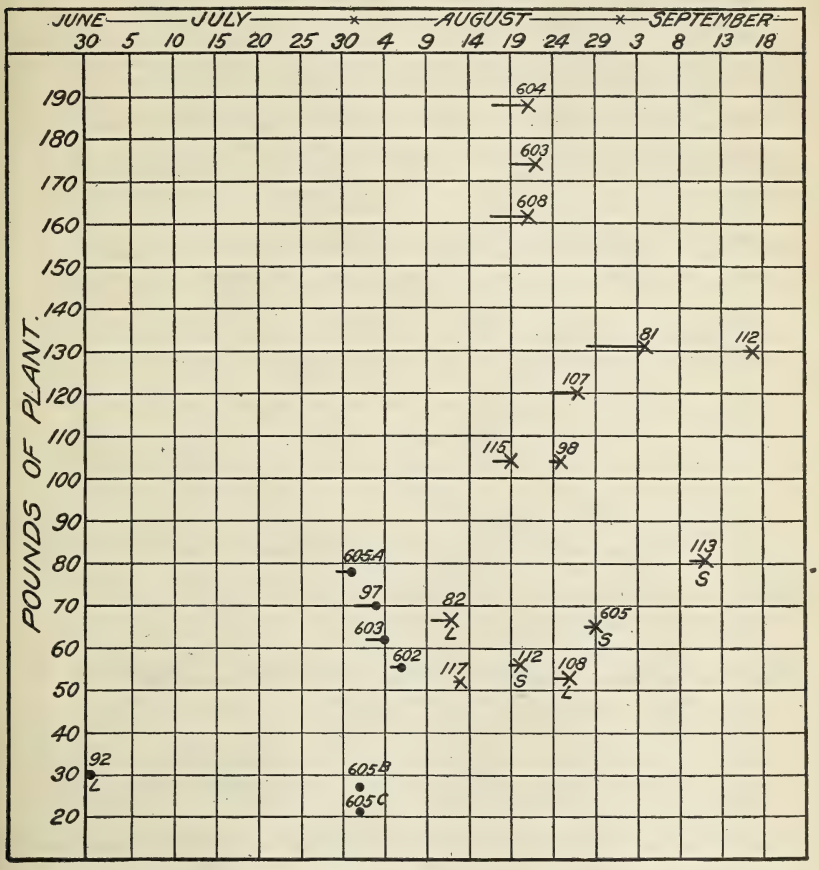

FIG. 6.—Chart of feeding of *Delphinium barbeyi* to cattle experimentally poisoned in 1909, showing dates, quantities fed, and duration of feeding. ● indicates plant collected near station; × indicates plant collected at Kebler Pass about 1,000 feet higher than the station; those marked *L* received leaves and stems; those marked *S* received seeds and the pods and stems bearing them; all the others received the whole top of the plant. The short horizontal line indicates duration of feeding. The weights of plant are given per thousand pounds of animal.

produce poisoning in 1909 varied from 30 pounds in the case of No. 92 to 188 in the case of No. 604. In 1910 the quantities varied from 30.4 pounds in the case of No. 98 to 280.8 pounds in the case of No. 625, while with the *Delphinium menziesii* the quantities varied from 62.2 pounds in the case of No. 113 to 116.5 pounds in the case of No. 82. In 1911 only *Delphinium barbeyi* was fed and the quantity necessary to produce poisoning varied from 34.7 pounds in the case

of No. 635 to 93.3 pounds in the case of No. 643. The averages of these cases, however, are very striking. The cases of 1909 averaged

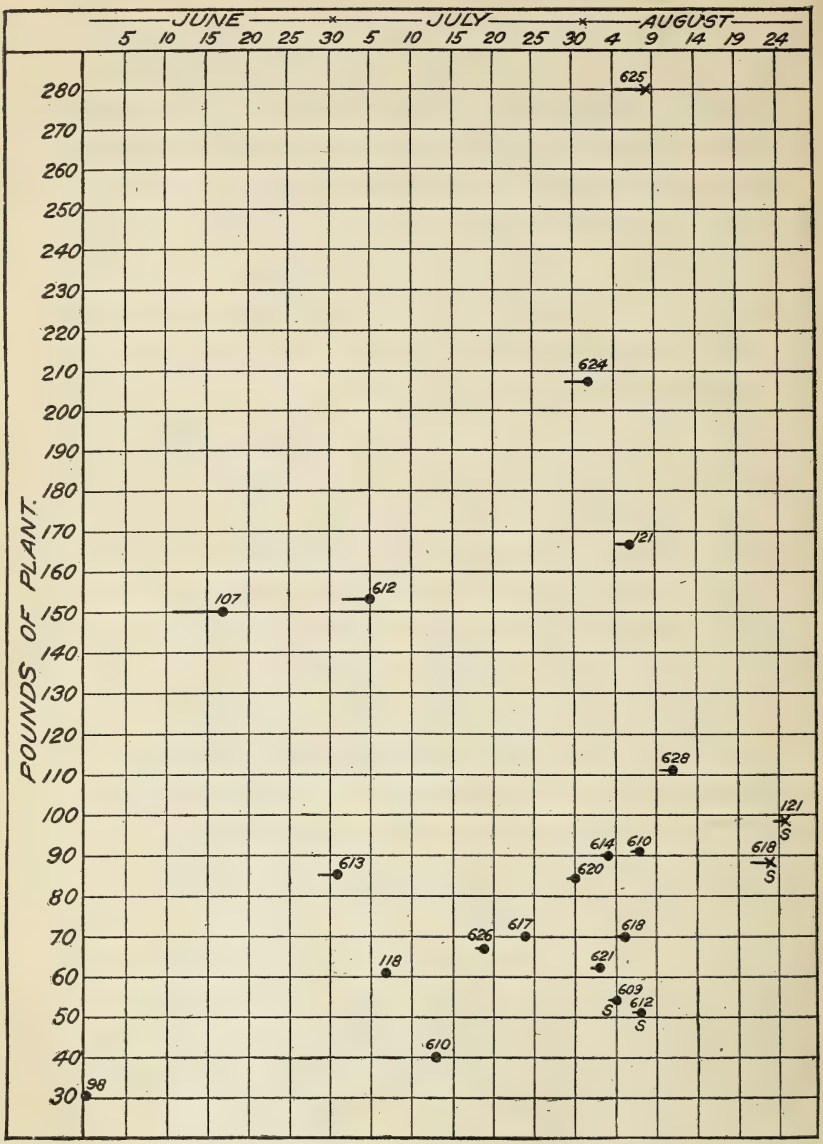

FIG. 7.—Chart of feeding of *Delphinium barbeyi* to cattle experimentally poisoned in 1910, showing dates, quantities fed, and duration of feeding. ● indicates plant collected near station; × indicates plant collected at Kebler Pass about 1,000 feet higher than the station; those marked *L* received leaves and stems; those marked *S* received seeds and the pods and stems bearing them; all the others received the whole top of the plant. The short horizontal line indicates duration of feeding. The weights of plant are given per thousand pounds of animal.

92 pounds; the *Delphinium barbeyi* cases of 1910 averaged 100.4 pounds, while the *Delphinium menziesii* feeding of 1910 averaged

95.8 pounds. The cases of 1911, all being of *Delphinium barbeyi* poisoning, averaged 63.3 pounds.

It was the impression among the observers at the station during the first two seasons that about one-tenth the weight of the animal was the toxic dose, and it is certainly rather remarkable that the averages come so close to that quantity. A careful study of the cases of the three seasons, however, shows not only that in the average case this is an overestimate, but that there are two factors which profoundly modify the quantity necessary to produce poisoning in individual cases. One factor, the seasonal variation in the toxicity of the plants, is discussed under a special heading on page 75. The second factor is the length of time during which the plant was fed. This is indicated in charts 11 to 14, and it will be noted that in general the size of the toxic dose increases with the time during which the animal is fed. This is shown in a striking way in the animals poisoned by *Delphinium barbeyi* in 1909. After tabulating the number of days of feeding and the quantities fed, and making averages of the cases, it was found that of the animals poisoned by 1 day's feeding, the average quantity was 53.2 pounds; of those poisoned by 2 days' feeding, 82.1 pounds; of 3 days' feeding, 133.7 pounds, and of 4 days' feeding, 160.1 pounds. The averages for the other two years show the same thing but not so clearly, as the seasonal variation in

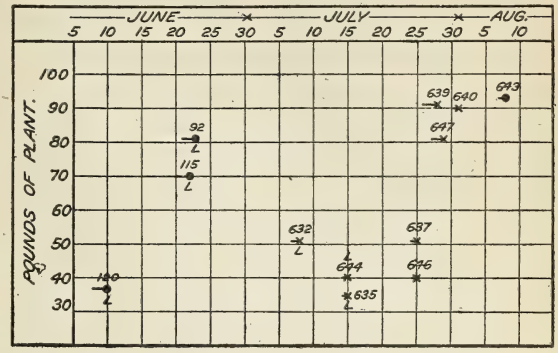

FIG. 8.—Chart of feeding *Delphinium barbeyi* to cattle experimentally poisoned in 1911, showing the dates, quantities fed, and duration of feeding. ● indicates plants collected near station; × indicates plants collected at Kebler Pass about 1,000 feet higher than the station; those marked *L* received leaves; the others were fed the whole top of the plant.

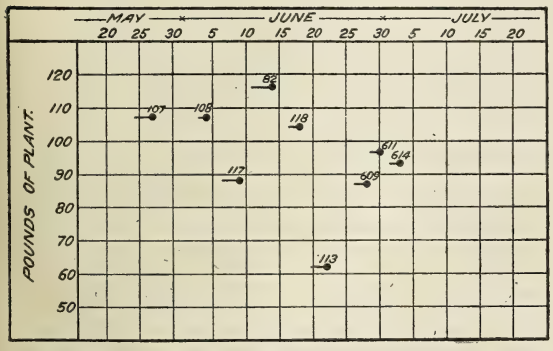

FIG. 9.—Chart of feeding of *Delphinium menziesii* to cattle experimentally poisoned in 1910, showing dates, quantities fed, and duration of feeding. The short horizontal line indicates duration of feeding. The weights of plant are given per thousand pounds of animal.

toxicity plays a more important part in those years. The average toxic dose for 1 day's feeding in 1910 was 54.9 pounds, and in 1911 it was 69.5 pounds. It thus appears that, in the general average of cases, cattle weighing 1,000 pounds will be poisoned if they eat as much as 60 pounds in one day. This quantity varies, however, within wide limits, in one case being as low as 30 pounds, and at the other extreme as high as 93.3 pounds.

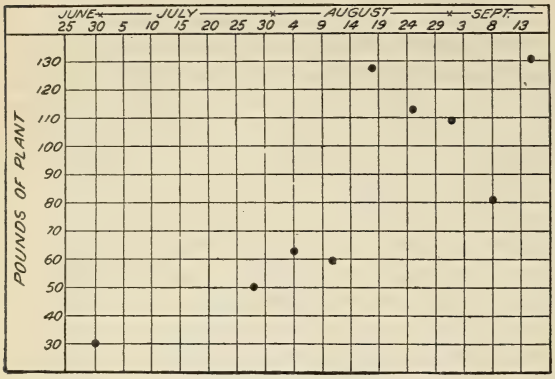

FIG. 10.—Chart of feeding of *Delphinium barbeyi* to cattle experimentally poisoned in 1909 based on weekly averages. The weights of plant are given per thousand pounds of animal.

A tabulation of the quantities eaten the first day by animals poisoned in 2, 3, or 4 days shows that few exceeded the toxic limit; of 15 cases in 1909, No. 115 ate 37 pounds, No. 98 ate 58.16 pounds, and No. 112 ate 56.5 pounds. Of 15 cases in 1910, No. 612 ate 43 pounds, No. 610 ate 36 pounds, and No. 121 ate 38 pounds, while in 1911, of 6 cases, No. 639 ate 62.2 pounds and No. 647 ate 46 pounds. It will be noticed that only one of these exceeded the average quantity which poisons in 1 day's feeding, but that all exceeded the minimum.

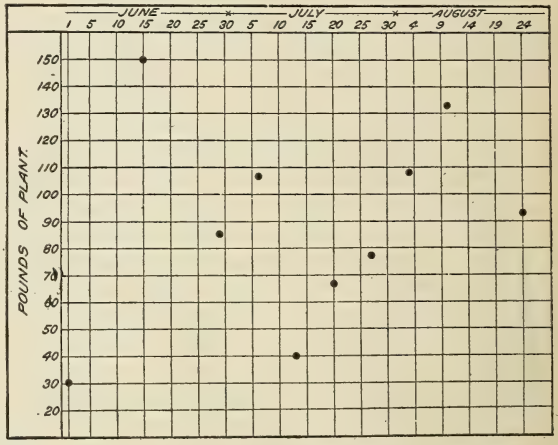

FIG. 11.—Chart of feeding of *Delphinium barbeyi* to cattle experimentally poisoned in 1910 based on weekly averages. The weights of plant are given per thousand pounds of animal.

While some of the differences in the toxic dose can be explained by seasonal differences in the plants and the duration of feeding, many remained unexplained. These differences, under apparently the same conditions, are shown in cases 637, 646, 639, 647, and 640 of 1911. All these animals were fed between July 25 and July 31, with the following

results: No. 637 was poisoned in 1 day by 51 pounds per 1,000 pounds of weight; No. 646, by 40 pounds; No. 640, by 90 pounds; No. 639 was poisoned in 2 days by 91.1 pounds; and No. 647, by 81.1 pounds. These differences are made more striking when we find that No. 639 ate 62.2 pounds the first day, and No. 647 ate 46 pounds. All these animals were of approximately the same age, treated in the same way with larkspur gathered from the same place, and all were fed within 6 days. The difference may be due in part to the condition of the animals when receiving the plant, for it is reasonable to assume that the rapidity of absorption may be affected by the condition of the alimentary canal and its contents. The condition of the excreting glands, too, may profoundly modify the toxic effect of the plants. Other minor factors doubtless come into play, which may be grouped together under the general term "the varying susceptibility of the individual."

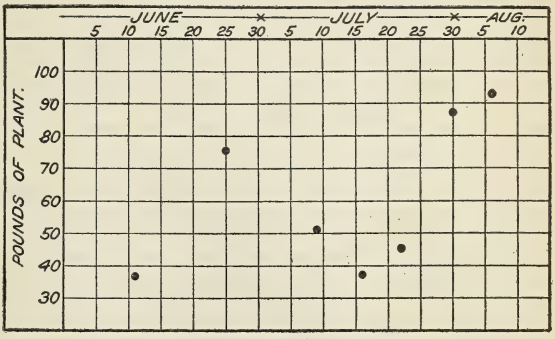

FIG. 12.—Chart of feeding *Delphinium barbeyi* to cattle experimentally poisoned in 1911, based on weekly averages. The weights of plant are given per thousand pounds of animal.

In this connection it may be noted that apparently rumination did not necessarily precede intoxication. While complete notes were not kept on this subject, it was definitely known that some of the animals which were poisoned in a short time did not ruminate at all. The minimum toxic dose, then, is about 30 pounds, and the average of the three seasons about 84 pounds, with a maximum of 280 pounds. This maximum, of course, would run to infinity late in the season. In the practical handling of cattle it is dangerous for an animal to eat more than 3 per cent of its weight in one day, although it may eat two or three times as much before showing signs of intoxication.

The figures, as given above, in regard to the toxic dose apply to *Delphinium barbeyi* and *Delphinium menziesii*, and it is interesting also to note that the quantity necessary to produce poisoning in the case of *Delphinium menziesii* does not differ materially from the quantity in the case of *Delphinium barbeyi*. In the single experiment with *Delphinium robustum* 40 pounds per 1,000 pounds of weight of the animal produced poisonous effects. Inasmuch as this feeding was rather late in the season, this single experiment would indicate that *Delphinium robustum* might be rather more poisonous than the two species experimented with at Mount Carbon. It is

not safe, however, to draw any definite inference in regard to this. The toxic dose in the experiments with *Delphinium cucullatum* varied from 22.7 pounds to 49 pounds. This apparently indicates a greater toxicity for this species than for the Colorado larkspurs. The experiments were few in number, however, and all taken during the time of probable maximum toxicity of the plant, and it seems likely that a wider experience would show greater conformity to the standard of the Colorado plants.

It is somewhat surprising to notice how great a quantity of larkspur must be eaten in most cases before poisonous effects are produced, and this fact may perhaps be the explanation of the cases which are frequently recorded of the passing of succeeding herds of animals over the same poisonous area, some being poisoned and others going without any harm whatever. It seems very probable that the animals showing the symptoms of poisoning may have come to these areas when particularly hungry and that individuals on this account may have eaten large quantities of the poisonous weed. It is well known that a ruminant when very hungry will eat enormous amounts of material which attracts it. It is also well known that under these conditions animals are more apt to take the plants which are most prominent, and if the larkspurs were more conspicuous than other forage plants it is very probable that the animal under such conditions would eat an unusual quantity and consequently suffer. The practical inference from this is that in handling cattle care should be taken not to drive them over a supposed poisonous area when they are particularly hungry. On this account it would doubtless be better to make the drive over such an area in the afternoon rather than in the morning. It will be noted, too, that the quantity which may be poisonous varies within very wide limits, and that an animal may suffer from eating not more than 25 or 30 pounds. Perhaps special emphasis should be placed upon the fact that the toxic dose is quite large. The larkspurs are not violently poisonous plants and may be eaten in quite large quantities with no bad results. Because a region contains some larkspurs it is not necessarily a dangerous locality for grazing. The region is dangerous only when the plants are present in considerable numbers or when there is a lack of other forage so that the cattle eat the larkspur in large quantities. *Delphinium menziesii* in some localities is so scattered that it can do no harm. This is true of areas in southern Utah. While *Delphinium bicolor*, the low larkspur which is characteristic of the region about the experiment station at Greycliff, undoubtedly has the same poisonous properties as the other larkspurs, it does not grow in that region in sufficient abundance to cause any harm. It occurs in scattered groups of a few plants and it would be impossible for cattle to get enough in grazing to produce intoxica-

tion. In fact, from what is known of the distribution of *Delphinium bicolor* it seems probable to the authors that this species is of no economic importance in causing losses of stock. It certainly does not poison sheep and it is highly improbable that it ever grows in sufficient abundance to be dangerous to cattle.

POST-MORTEM FEATURES OF LARKSPUR POISONING.

During the season of 1909 three autopsies were made upon the station experimental animals and three upon others that were supposed to have died of larkspur poisoning. In 1910 nine autopsies were made on animals that died at the station, and in 1911 three. Generally speaking, as has been noted elsewhere, if animals found dead upon the range are lying upon uneven ground, the head will be found lower than the rest of the body. This was true also of the animals that died in the corrals, and is probably explained by the fact that as the animals throw themselves about they get their heads lower and are unable to turn themselves back.

Generally, too, the animal dying upon the range is found very much bloated. It is very difficult to determine the post-mortem condition of range animals, as it is seldom possible to make autopsies immediately after death, and as the number of animals autopsied at the station was small the facts observed can not be supposed to demonstrate conclusively the detailed conditions of larkspur poisoning.

In nearly all cases the heart was found in diastole and filled with blood. Commonly, the walls of the heart were more or less congested and frequently with petechiæ. The peripheral veins and venous system of the abdomen were found congested. In stripping the skin from the animal it was usual to find the veins immediately beneath the skin very much swollen. The lungs were congested, and the kidneys acutely congested. There was generally a hyperemic condition of the central nervous system, as would be expected from the general condition of the circulatory organs. Commonly the inner walls of the trachea and sometimes of the bronchi were very deeply congested. Inflammation was almost invariably present in the rumen near the esophageal opening. In some cases the walls of the second and third stomach were inflamed and in practically all cases the pyloric end of the fourth stomach. This inflammation extended in greater or less degree through the duodenum, jejunum, and ileum. In three cases the colon was inflamed. In five cases the wall of the cecum was inflamed, and in most cases the walls of the rectum.

To summarize the noticeable points brought out by the post-mortem examinations of these animals, there was marked inflammation in all parts of the alimentary canal, marked congestion of

the kidneys, and distinct congestion of the walls of the heart, associated with a general congestion of the peripheral circulation.

TOXICITY OF DIFFERENT PARTS OF THE PLANT.

In the course of the experiments careful notes were made with regard to the part of the plant fed to the animals. Some animals were fed leaves and stems; others leaves, stems, and flowers; others the tops with the seed; and, in the case of *Delphinium menziesii* and *Delphinium andersonii*, some were fed the roots alone.

There is a widespread belief among the stockmen of Colorado that the roots of *Delphinium menziesii* are much more poisonous than other parts of the plant. It is said that cattle are much more likely to be poisoned after a rain, when they can pull up the plants by the roots and devour a large quantity of the latter. In the summer of 1909 special attention was paid to the feeding of roots to the cattle. Two animals—Nos. 92 and 117—were fed roots alone of *Delphinium menziesii*. No. 92, in 2 days, ate an equivalent of 2.47 pounds per 1,000 pounds of weight, and No. 117, in 1 day, ate 2.1 pounds of roots without any symptoms of poisoning. These quantities, to be sure, were not very large; but it is highly improbable that an animal upon the range would ever be able to consume as much. The stem of *Delphinium menziesii* is quite brittle and, while it is entirely possible to pull up the roots by the stems while the soil is moist, the larger part of them, as was proved by experiment, will break, and it is improbable that cattle in their grazing will get any considerable number of roots. These experiments would seem to prove that the roots of *Delphinium menziesii* are not violently toxic. The roots of *Delphinium barbeyi* are long and tough and are never pulled up by stock, so that for grazing they need not be considered. The feeding experiments with *Delphinium menziesii* throughout the season of 1910 were of the whole plant, and there was no reason to think that the roots were especially toxic. In the experimental feeding of the roots of *Delphinium andersonii*, given in detail on page 58, only sheep were used, so no results were reached as to the comparative toxicity of different parts of the plant, as there is no evidence that sheep are poisoned by any part of the plant. The experiment was significant as indicating that in all probability sheep are not injured by the roots of this plant.

The charts (figs. 6, 7, 8, 9, and 10) for the feeding of both *Delphinium barbeyi* and *Delphinium menziesii* show quite clearly the greater toxicity of the seeds. It will be noticed from the charts that in the feeding of plants at the time when seeds were present a smaller quantity was necessary in order to produce symptoms of poisoning. In this connection, the case of heifer No. 633 is especially interesting. This animal was found dead in the pasture September 2, 1911.

Although *Delphinium barbeyi* was common in the pasture, no trouble had been experienced from this source, probably because there was an abundance of good feed. Moreover, none of the experimentally fed animals had been poisoned since August 8, on account of the diminished toxicity of the plants. The autopsy showed that No. 633 had died of asphyxia, as it had vomited, and the stomach contents were found in the larynx and trachea. As the animal had been dead for two or three days, the autopsy was unsatisfactory, but, so far as it could be made, showed conditions typical of larkspur poisoning. A careful examination of the contents of the rumen demonstrated the presence of a large amount of stems and seeds of *Delphinium barbeyi*. This, then, was clearly a case of larkspur poisoning in which the seeds were the most important factor, for it was too late in the season for the leaves to produce poisoning.

AGE OF PLANTS AS AFFECTING TOXICITY.

From a careful examination of the charts for the feeding of *Delphinium barbeyi* and *Delphinium menziesii* certain facts are brought out quite clearly in regard to seasonal changes in toxicity. If an average curve were made for the charts of *Delphinium barbeyi* feeding in 1909, 1910, and 1911 (figs. 6, 7, 8, 10, and 11), it would be found that the quantity necessary to produce poisoning increases progressively from the first of the season until the time when seeds are formed in the plants. Taking into account the length of time during which the plant was given in individual cases, the apparently aberrant cases of very large quantities in these years are easily explained, as, in those cases, by reason of the prolonged feeding, there was more or less elimination of the poison.

It is a striking fact that the smallest quantity needed to produce poisoning was in the earliest cases. It seems quite clear that *Delphinium barbeyi* progressively loses toxicity after blossoming until the time when the seeds are formed. At this time the leaves and stems are not particularly toxic and if the seeds were disregarded, the curve would indicate diminished toxicity from early in the season until the middle or last of August, at which time on the Colorado ranges the plant becomes perfectly harmless.

As a matter of fact, stock on the range do not eat the seeds of *Delphinium barbeyi* to any extent, so that the fact that the seeds are especially toxic has little practical bearing so far as the stockmen are concerned. It may be stated as a general fact that after the middle or latter part of August, depending upon the season, *Delphinium barbeyi* ceases to be poisonous, and under ordinary range conditions in Colorado few cases of poisoning occur after the middle of July. Not only does it cease to be injurious, but it has been noticed that

late in the season during the month of September the leaves of *Delphinium barbeyi* are eaten by stock with great apparent eagerness. Before the season is concluded, where a range is grazed with any thoroughness, nearly all the leaves of *Delphinium barbeyi* will be stripped from the stems by the grazing cattle and eaten with no resulting harm.

The chart for *Delphinium menziesii*, figure 9, determined by the experiments of 1910, would seem to indicate that the quantity necessary to poison stock grows smaller as the season progresses. This probably is explained by the fact that in the latter part of June many of the plants have formed seed and that these seed pods were eaten by the cattle. If the plant has greater toxicity in the latter part of the season than in the earlier, as this chart would seem to indicate, it is doubtless explained in this way, for the seeds are formed in *Delphinium menziesii* while the leaves are still more or less green and doubtless attractive to a grazing animal.

The principal inferences from these facts in regard to the variation of toxicity with the age of the plant may be summed up as follows:

First, *Delphinium menziesii* is poisonous during the whole period of the life of the plant. Immediately upon the formation of the seed, the plant withers and disappears, so that it no longer is a factor in poisoning. If *Delphinium menziesii* does more harm in the early season than in the latter period of its existence, it must be due to the fact that, because of the poorer feed earlier in the season, cattle may eat more of it than they do later when the grasses have sprung up.

Second, *Delphinium barbeyi* in Colorado is poisonous from early spring until the middle or last of August, its toxicity after blossoming gradually diminishing until it entirely disappears and the plant can be eaten with impunity by cattle. It would appear that it is vastly more toxic early in the season and without doubt it is in the month of June that the most harm is done by this plant. The fact of the great toxicity of the seeds has little practical importance because cattle rarely feed upon them. So far as inferences may be drawn from a somewhat limited experience it would appear that *Delphinium cucullatum* varies in its toxicity as does *Delphinium barbeyi*.

Investigations in the Sierras, where the common larkspur is *Delphinium glaucum*, show a somewhat different condition from that noted in Colorado. Here the snowfall is very heavy and the snow does not disappear in some localities until very late in the season, making the period of blossoming late. Larkspurs may be in blossom as late as September, and the period of possible poisoning of cattle is extended through nearly the whole grazing season.

It should be borne in mind also that in any given region, climatic conditions vary. In a dry, hot season the larkspurs will ripen earlier, while in a cold, wet season the time of blossoming and forming of seed may be much delayed.

Referring to the work of Loy, Heyl, and Hepner, which is noticed on page 11, it will be seen that their results in regard to the toxicity of different parts of the plant correspond fairly well to the results obtained in the field experimentation. It may be noted that the large content of alkaloid in the leaf and stem of *Delphinium geyeri* as compared with the other species may be accounted for by the fact that the plant was collected early in the season before blossoming, at the time when it might be expected to be more toxic, while the *Delphinium glaucum* was collected at the full maturity of the plant and very likely at a time when the toxicity was beginning to diminish.

ANTIDOTAL TREATMENT OF CASES OF LARKSPUR POISONING.

The early treatment of larkspur poisoning at the Mount Carbon station was based upon the recommendations in the literature of the subject. Wilcox, 1897, page 45, recommends the use of atropin sulphate, stating that he had had good results with sheep in Montana. Chesnut and Wilcox, 1901, pages 72 and 80, recommend atropin for counteracting the physiological effects, and suggest that alcoholic stimulants and ammonia can be used to advantage. They recommend also permanganate of potassium and sulphate of aluminium. Crawford, 1907, pages 9 and 10, states that poisoning takes place more quickly when elimination is interfered with, as, for example, by tieing the ureter of the animal experimented upon. It seemed best, therefore, in the experimental work at Mount Carbon to make trial of atropin, potassium permanganate, and caffein sodio-benzoate. The latter substance was used partly because it is a heart stimulant and partly because it is a diuretic, on the assumption that stimulation of the kidneys might aid in the elimination of the poison. In several cases during the first season's work at Mount Carbon these remedies were used, and while all of the animals to which the remedies were given recovered, there was reason to think that none of the remedial measures were especially effective. On comparison of the animals treated with those not treated, it could not be shown that there were any advantageous effects from the administration of these remedies.

Reference may be made here to the experiments detailed in pages 41 to 43 of United States Department of Agriculture Bulletin No. 125, "Zygadenus, or Death Camas," in which it is shown that good results can not be reasonably expected from an antidotal remedy like potassium permanganate, given per os to a ruminant, inasmuch as the antidote is not likely to come in contact with any considerable

quantity of the poisonous substance unless it is given in many doses repeated at very frequent intervals.

It was noticed early in the work of 1909 that all the poisoned animals were very constipated, and the question was raised whether the removal of this condition might not either prevent the poisoning or predispose the animals to recovery. Cowboys upon the range have remarked that whenever animals commence to defecate recovery is assured. Therefore if the animals were so treated as to keep up a free movement of the bowels, it might be possible to prevent the poisonous action of the larkspur. To test this, No. 602 was brought into the corral on September 8, 1909, for experimental feeding. Feeding of *Delphinium barbeyi* was commenced on September 9, using the leaves, stems, and fruit of material that had been collected at Kebler Pass. Although this material was mature, it was green and fresh. Feeding was continued to September 16. During this time the animal, which weighed about 450 pounds, ate 388.25 pounds of the plant, or, on the basis of 1,000 pounds of weight, 862.8 pounds. On September 9, 10, 11, 12, 13, 15, and 16 she received 4 ounces of magnesium sulphate in the drinking water. In spite of the large quantity of larkspur eaten the animal showed not the slightest effect of poisoning. The bowels were kept rather more loose than normal. Inasmuch as the general results of the experimental work show that the larkspur as it grows older loses much of its toxicity, the question was raised whether the failure to poison this animal was not due to the fact that the larkspur was old and had perhaps lost some of its poisonous properties. In order to test this No. 112 was brought into the corrals on September 15, and feeding was commenced on September 16 of material obtained from the same place as that fed to No. 602. She was fed during September 16 and 17 79½ pounds, or, on the basis of 1,000 pounds of weight, 130 pounds. At 5.35 p. m. on September 17 she was found down in the corrals. At 5.38 she was disgorging material from the rumen, this material consisting of larkspur and water, part of it passing up through the nostrils and interfering with her breathing. At 5.42 she was raised up in order that the trachea might be less likely to be filled with the vomited material. She was hardly able to hold up her head. There was some twitching of the flank muscles and the muscles of the forelegs. Respiration at this time was very slow and shallow. The pulse could not be found at all. At 5.48 she was dead. This animal during the feeding was very much constipated. She received larkspur from the same localities as that fed to No. 602, and the material was in practically the same condition. It should be noted, too, that not only did No. 602 receive a much larger total quantity of larkspur, but the daily feeding also was very much

larger. On one day this animal received almost twice as much as was given to No. 112 on the second day when it became ill.

While these two cases can not be considered as furnishing positive proof that the administration of magnesium sulphate will prevent the action of larkspur, the results were very significant.

In connection with this case, comparisons may be made with some others. No. 606, a heifer, weighing about 450 pounds, belonging to Otis Moore, was fed, between August 28 and September 6, 195 pounds of *Delphinium barbeyi*, or, on the basis of 1,000 pounds of weight, 434.8 pounds. Part of this material was collected at Kebler Pass and was green. A smaller part, about 50 pounds, was collected near the station and was older and drier. This feeding was of leaves and stems without the seeds. She was given 4 ounces of magnesium sulphate in the drinking water on August 30 and September 3. No poisonous effects were noticed.

At the same time, August 28 and 29, No. 605 was fed 29½ pounds, or, on the basis of 1,000 pounds weight, 66.5 pounds, and became sick. The material fed was of stems and seeds of *Delphinium barbeyi*. It should be borne in mind, however, in comparing Nos. 605 and 606, that the seeds are more toxic than the leaves and stems, as has been shown elsewhere, and that it is possible the result in the case of No. 605 may have been caused by the larger number of seeds in the feeding.

With this, however, may be compared No. 98, which, between September 18 and 25, received 357.25 pounds, or, on the basis of 1,000 pounds' weight, 776.6 pounds of *Delphinium barbeyi*, collected at Kebler Pass. This material included not only stems and leaves, but the seeds. The animal ate a very large proportion of its own weight of larkspur. Four ounces of magnesium sulphate in its drinking water were given every day between September 18 and 25, inclusive, the effect of this being to keep the action of the bowels in very nearly a normal condition. The animal was not affected at all by the poisonous material eaten.

Summing up these cases, then, it would appear that it is very probable that the injurious effects of larkspur eating might not appear if means were taken to produce free movement of the bowels in the animals feeding upon the plant, and it indicates also that if some remedy could be used which, in the beginning of the poisoning, would quickly stimulate the intestinal excretion it might serve to save the lives of the animals.

Inasmuch as the work of 1909 at the Mount Carbon station brought out very clearly the fact that one of the most prominent symptoms connected with larkspur poisoning was constipation, and also showed very clearly that death resulted primarily from respiratory paralysis,

in planning for the remedial work of 1910 it seemed wise to use substances which would probably counteract these most pronounced symptoms. It was at first thought that some combination might be made with barium chlorid, using the barium chlorid for the purpose of getting a quick evacuation of the intestines, combining with it caffein or digitalis to relieve the depressing effect which barium has upon the heart and adding strychnin to serve as a respiratory stimulant. Tablets were prepared of various combinations for the summer's work.

One case of *Delphinium menziesii* poisoning was treated with barium chlorid, caffein, sodio-benzoate, and strychnin nitrate, and died. One case of *Delphinium barbeyi* was treated with the same combination and died. It was not clear, therefore, that there were any beneficial results from this treatment, and as it was found difficult to handle the combination without hot water for solution it was abandoned as impracticable for field use.

A hypodermic injection was used of physostigmin salicylate, pilocarpin hydrochlorid, and strychnin sulphate. This combination dissolves very readily and can be used in a comparatively small amount of water. The treatment was used in 32 cases of larkspur poisoning with a total of 4 deaths. One fatal case was known to be due to an overdose of strychnin and two received too small a dose of physostigmin. One case died, apparently, in spite of the remedy. Fifteen were allowed to go without treatment, and of these 6 died. This seems to make a good showing for the remedy, although, of course, too much stress must not be put on the statistical results of a comparatively small number of cases. It is presumed that probably a larger proportion of range animals would die than of corral-fed cases, for the latter, even if no remedy was given, are cared for and put in a favorable position for recovery.

Excluding the animal killed by strychnin and the 2 receiving too small a dose, there was only 1 death in 29 treated cases; in other words, there was 96.54 per cent of recoveries. While this percentage might not hold in a larger number of cases, there is good reason to believe that most cases of larkspur poisoning may be cured if this treatment can be applied promptly.

In comparing the effects obtained in the different cases it was found that the best results in animals weighing 500 to 600 pounds were reached by using the following formula of this remedy:

Physostigmin salicylate	1 grain.
Pilocarpin hydrochlorid	2 grains.
Strychnin sulphate	½ grain.

As much as 1 grain of strychnin was used in some cases, but it seems probable that this is too much. There was little doubt that an overdose was given to No. 613, a fatal case of *Delphinium barbeyi*

poisoning in 1910, as there were distinct symptoms of strychnin poisoning. Smaller doses were tried with some of the cases of 1911, but they were less effective and the two fatal cases in this season, when this remedy was used, are considered as due to the use of an insufficient amount of the remedy. It is possible that a heavier dosage of physostigmin salicylate and pilocarpin hydrochlorid might be used, but experience seemed to show that the pain connected with the more rapid action of this remedy more than counterbalanced its advantage. The results of the summers of 1910 and 1911 appeared to show quite conclusively that the hypodermic injection of this combination would aid in the recovery of most animals. The attempt was made to use arecolin in place of the physostigmin and pilocarpin but the results were very unsatisfactory.

It was found that a distinct benefit resulted from the use of hypodermic injections of 20 cubic centimeters or more of whisky or a corresponding amount of 50 per cent alcohol. This stimulant was given to tide over a time when the animal might otherwise collapse. It was not found desirable to give the whisky in all cases but only as the symptoms seemed to demand it.

In passing, perhaps a word should be said in regard to the ordinary remedy of bleeding used among the stockmen for larkspur poisoning. This was not attempted in the station work, because there seemed to be no good reason for the proceeding. It is barely possible that at the critical stage of larkspur poisoning, with the heart about to stop, bleeding might stimulate it to further action. It was not found, however, in the station experiments that the symptoms at any time definitely indicated this as a desirable measure. Indiscriminate bleeding for larkspur poisoning is probably worse than useless and does much more harm than good. Among stockmen the claim is frequently made that 50 per cent of the sick cases may be saved by bleeding. It may be questioned whether this number might not recover without any treatment. Dr. Sanford, of Gunnison, Colo., a physician of long and successful experience in a stock country, states that he has bled a large number of animals poisoned by larkspur and has no evidence of beneficial results.

Bleeding is the common remedy used by stock people for many of the ills affecting their animals, and is considered especially efficacious in cases of illness resulting from eating poisonous plants. While it did not seem worth while to test it out in the larkspur poisoning of cattle, it was used experimentally with sheep poisoned by Zygadenus (death camas), as stated in Bulletin 125, with no benefit.

Summarizing, then, the work of the station upon remedies, no definite advantageous results were obtained with potassium permanga-

nate, atropin, or the combination of barium chlorid with caffein, sodio-benzoate and strychnin. The combination of physostigmin salicylate, pilocarpin hydrochlorid, and strychnin sulphate, used hypodermically, and supplemented as symptoms demand by hypodermic injections of whisky or dilute alcohol, would seem in the majority of cases to produce beneficial effects. These remedies can be easily administered by stockmen upon the range, as they can be carried in solution in small compass and administered by the hypodermic syringe, with the use of which most stockmen are familiar. It can not be too strongly stated that when cattle fall from larkspur poisoning no attempt should be made to get them upon their feet, or, if they get upon their feet themselves, care must be taken that they should not be hurried under any circumstances. Many of the animals when poisoned, if allowed to lie quietly with no other attention than to be turned so that the head will be higher than the rest of the body, will recover.

As has been stated elsewhere, bloating seldom occurs in cases of larkspur poisoning. If it does, it should be relieved by paunching, and every stockman should be provided with a trocar to perform this operation.

METHODS OF PREVENTING LARKSPUR POISONING.

It is recognized that under ordinary range conditions many cases of larkspur poisoning occur which can not be prevented. The cattle are not under direct observation and may not be seen for weeks or months, and the first intimation of trouble is when the rider, in going over the range, finds bodies of animals that may have died long before. There is no opportunity to apply a remedy. It is possible, however, to save many cattle by proper handling in accordance with the conditions of the ranges upon which they are grazed.

From the fact that the low larkspur dies early in July and ceases to be a factor in poisoning, it is very evident that if the cattle can be kept away from this plant until about July 1 there probably will be no fatalities. This has been recognized very generally by the stockmen. In some localities on the western slope of the Rocky Mountains in Colorado "riding for poison" is a regular business among the stockmen during the month of June. By this "riding" the cattle are kept below the poisonous area until after the plants blossom. In some localities, also, through the instrumentality of the Forest Service, drift fences have been erected for the same purposes.

There seems to be no question that if cattle can be kept away from the areas of low larkspur until the plant matures there will be no losses, but if they are permitted to graze freely upon these areas loss is almost certain to occur. These losses, of course, will be greater

when the grasses are less conspicuous. Just so far as the larkspur is more evident than other forms of forage plants, it is sure to be eaten in larger quantities and will produce correspondingly greater harm.

The tall larkspur is especially dangerous in Colorado during the months of May and June. After it springs up in the early part of the season it grows in large tufts of rather attractive appearance and extends above the forage plants. It is at this time that it is most likely to be eaten by cattle. In narrow valleys where the larkspur is quite abundant, if cattle collect in the early part of the season to graze, they are almost certain to take a considerable quantity of the larkspur with more or less losses resulting. It is entirely feasible in many of these small canyons to clear out the major part of the larkspur and thus prevent poisoning, and it is definitely recommended that in such restricted areas the plant be dug out.

Experimental work carried out upon the range has shown that the larkspur can be killed by cutting the root 2 or 3 inches below the surface of the ground, and this has been done by the Forest Service in some localities on a somewhat large scale. Complete eradication of the plant, however, is impossible, and in many places it is economically unprofitable to dig it out. In some valleys it is so scattered among the willows that it is difficult to approach it, and on some ranges it is distributed so widely and in places so difficult of access that the expenditure of labor necessary to destroy the plant would exceed the value of the range. The practicability of digging out larkspur on any range depends upon the characteristics of that particular range, and can not be decided without a careful examination of local conditions.

It was found, while investigating the conditions of larkspur poisoning in the Sierras, that in many especially harmful regions the heavy growth of larkspur is confined to particular valleys, or, in some cases, to a very limited area in a valley. Some of these valleys can be easily fenced off and used for horses rather than for cattle, and the small isolated areas can be cleared of most of the larkspur at a small expenditure of time and money.

When cattle are driven hurriedly from one range to another they are much more apt to become poisoned, as it is well known that hungry cattle when hurried along will eat the most conspicuous plants, and under such circumstances quite large losses may occur. It is evident, then, that in handling cattle in areas where the tall larkspur is abundant, particularly early in the season, great care should be taken that they should not come upon these areas when they are especially hungry. The subject of the proper handling of range animals in order to avoid poisoning is treated more specifically

in Farmers' Bulletin No. 720, Prevention of Losses of Live Stock from Plant Poisoning.

After the plant has matured, as has been shown elsewhere, its toxicity diminishes, and cattle, finding at the same time an abundance of other more attractive feed, eat very much less of the larkspur so that the danger of poisoning is very slight, and in the fall, after the plant begins to dry, cattle may and do eat it in large quantities with impunity.

It is generally considered by stockmen that poisoning is more likely to occur immediately after a rain, or even when the plants are wet with dew. There seems to be no reasonable explanation of the supposed fact of the greater toxicity of the plant when wet. It seems possible, however, when cattle are feeding hastily in a larkspur area after a rain, that rather than thrust their heads and faces into the wet grass they may eat more of the higher plants; in this way they would consume more of the larkspur and consequently become poisoned. Cattle, too, in the time of a storm gather together in the valleys and under trees where larkspur is very abundant, and doubtless eat more of it on this account.

Probably, also, when cattle are handled upon a supposed poisonous area it would aid somewhat in preventing loss if pains were taken to make sure that none of them were constipated. This probably could be accomplished, where cattle are watered at specific places, by the use of a small amount of magnesium sulphate or sodium sulphate in the drinking water.

GENERAL SUMMARY.

1. The larkspurs from very ancient times have been recognized as poisonous plants, but complaints of stock poisoning by these plants have been confined almost entirely to the mountain ranges of western North America, where heavy losses have been reported, especially among cattle.

2. It is rarely possible to recognize macroscopically larkspur material in the stomach contents of cattle. By means of microscopic sections of stems, however, not only can Delphinium be distinguished from other plants but groups of the genus can be distinguished from each other. The genus falls into six different types of stem structure.

3. Experimental feeding of larkspur was carried on for three seasons at Mount Carbon, in Gunnison County, Colo. In this work four species of Delphinium were used which have been identified as *Delphinium barbeyi*, *D. menziesii*, *D. andersonii*, and *D. robustum*. A large number of animals were used in this work, including horses, cattle, and sheep. Similar feeding experiments were conducted

during one season at Greycliff, Mont., on *Delphinium cucullatum* and *D. bicolor*.

4. These experiments showed that the larkspurs are poisonous to cattle and horses but not to sheep. Horses, however, in pastures or upon the range do not eat enough of the plants to produce any ill effects, so that losses of stock from larkspur poisoning are confined to cattle.

5. The low larkspurs are poisonous during the whole life of the plants, but inasmuch as they disappear early in July, cases of poisoning are confined to the months of May and June.

6. The tall larkspurs live through the summer season, appearing in early spring. They are most poisonous in their early stages. After blossoming the toxicity gradually diminishes and disappears and the plant dries up, although the seeds are very toxic. Most of the cases of poisoning in Colorado occur in May and June, with sporadic cases in July. In other localities where the larkspurs blossom later poisoning may occur as late as August or even September.

7. While definite feeding experiments have been performed upon only a few species of larkspur, it may be assumed, from the knowledge of plant poisoning upon the ranges, that other species have the same properties as those experimented upon and that feeding upon them produces the same results.

8. The experimental work and the autopsies showed a clearly defined line of symptoms and certain definite pathological results.

9. The feeding showed that there was no marked difference in toxicity between the different species of larkspurs and that the quantity necessary to produce effects varied within rather wide limits, but that, generally speaking, a quantity equal to at least 3 per cent of the weight of the animal was necessary to produce poisoning.

10. From somewhat extensive experimental work on antidotes it was found that beneficial results could be obtained by using, hypodermically, injections of physostigmin salicylate, pilocarpin hydrochlorid, and strychnin sulphate, followed by hypodermic injections of whisky when needed.

11. Poisoning upon the range may be prevented in some cases by digging up the tall larkspur when the greater number of plants is confined to comparatively limited areas. In other cases the handling of the cattle in such a way that they will not have an opportunity to feed upon the larkspur may prevent losses. In the case of *Delphinium menziesii* it is desirable that the cattle should be kept away from the ranges where this plant grows in abundance until about the 1st of July, when the plant dies. *D. barbeyi* loses its toxicity after blossoming, so that a range with this plant is safe for cattle in the late summer and fall. It should be remembered, however, that local

and climatic conditions may delay the time of blossoming, so that no arbitrary date can be given when a range is safe. *D. bicolor* probably never grows in sufficient quantities to be dangerous as a poisonous plant. Inasmuch as the experimental work seems to show quite conclusively that sheep may feed upon larkspurs with entire impunity it is desirable in some cases, where there is an especial abundance of larkspur, to use the ranges for sheep rather than for cattle or to combine sheep grazing and cattle grazing in such a manner as to keep the areas of low larkspur eaten down by the sheep.

LITERATURE CITED IN THIS PAPER.

Bessey, C. E.

1902. A preliminary account of the plants of Nebraska which are reputed to be poisonous, or are suspected of being so. Nebraska State Board of Agriculture, 16th Annual Report, 1901, pp. 95–129.

Beier.

1845. No title. Landwirtschaftliche Zeitung. Cited in Dammann, 1886, p. 841.

Blankinship, J. W.

1903. The loco and some other poisonous plants in Montana. Montana Agricultural Experiment Station Bulletin 45, pp. 93–95, 100, 101, fig. 6.

Boehm, R., and Serck, Julius.

1876. Beiträge zur Kenntniss der Alkaloide der Stephanskörner (Delphinium staphysagria). Archiv für experimentelle Pathologie und Pharmakologie, Bd. 5, pp. 311–328.

Cayrade, P.

1869. Sur l'action physiologique de la delphine. Journal de l'Anatomie et de la Physiologie, ann. 6, pp. 317–326.

Chesnut, V. K.

1898. Principal poisonous plants of the United States. U. S. Department of Agriculture, Division of Botany, Bulletin 20, pp. 23–26, fig. 9.

1898a. Thirty poisonous plants of the United States. U. S. Department of Agriculture, Farmers' Bulletin 86, pp. 11–13, fig. 6.

1899. Preliminary catalogue of plants poisonous to stock. U. S. Department of Agriculture, Bureau of Animal Industry, 15th Annual Report, 1898, pp. 400–401.

Chesnut, V. K., and Wilcox, E. V.

1901. The stock-poisoning plants of Montana; a preliminary report. U. S. Department of Agriculture, Division of Botany, Bulletin 26, pp. 65–80, pls. 2–5.

Crawford, A. C.

1907. The larkspurs as poisonous plants. U. S. Department of Agriculture, Bureau of Plant Industry, Bulletin 111, pp. 5–12, pl. 1.

Dammann, Carl.

1886. Die Gesundheitspflege der landwirtschaftlichen Haussäugethiere. Berlin. pp. 840–841, 1072.

Delafond, O.

1843. Traité sur la maladie de sang des bêtes à laine, Paris, pp. 172–174, 191, 194, 199.

Dioscorides.

De materia medica. Lib. 4, cap. 156.

Falck, P. C., and Rörig, C.

1852. Das Delphinin und das Pflanzengenus Delphinium. Archiv für physiologische Heilkunde, Jahrg. 11, pp. 528–546.

FROGGATT, W. W.
1900. Plague locusts. Agricultural Gazette of New South Wales, v. 11, p. 181.

GERLACH, A.
1845. Die Blutseuche der Schafe in Rücksicht der Ursachen, der Ansteckungsfähigkeit und der Vorbauung. Magazin für die gesammte Thierheilkunde, Jahrg. 11, p. 125.

GLOVER, G. H.
1906. Larkspur and other poisonous plants. Colorado Agricultural Experiment Station, Bulletin 113, pp. 10–19, pl. 1–3.

HAHN, L.
[1882.] Dictionnaire encyclopédique des sciences médicales, t. 26, pp. 523–543.

HALL, H. M., and YATES, H. S.
1915. Stock-poisoning plants of California. California Agricultural Experiment Station, Bulletin 249.

HEYL, GEORGE.
1903. Ueber Feinde der Haustiere in der Pflanzenwelt und ein giftiges Prinzip einiger Delphiniumarten (Delphocurarin). Süddeutscher Apotheker Zeitung, Band 43, Nos. 28, 29, 30.

HUTH, E.
1895. Monographie der Gattung Delphinium. Botanische Jahrbücher [Engler's], Bd. 20, pp. 322–499, pl. 6–8.

IRISH, P. H.
1889. Plants poisonous to stock. Oregon Agricultural Experiment Station, Bulletin 3, pp. 25–26.

JELLIFFE, S. E.
1899. Plant histology. Rusby, H. H., and Jelliffe, S. E. Morphology and histology of plants. New York. pt. 2, p. 339.

KNOWLES, M. E.
1897. Larkspur poisoning of sheep and cattle. First Annual Report of the Board of Sheep Commissioners of Montana, pp. 27–28.

KNOWLES, M. E., and STARZ, E.
[1897.] Larkspur poisoning of cattle and sheep. Montana State Veterinarian's Office, Circular 1.

LENFANT, C.
1897. Contribution à l'anatomie des renonculacées: le genre delphinium. Archives de l'Institut Botanique de l'Université de Liége, v. 1, 70 p., 11 pl.

LOY, S. K., HEYL, F. W., and HEPNER, F. E.
1913. Analysis of some Wyoming larkspurs, I. Twenty-third Annual Report, University of Wyoming Agricultural Experiment Station, pp. 73–79.

MACGREGOR, A.
1908. Delphinium poisoning in a cart mare. Veterinary Journal, London, v. 64 (n. s. v. 15), p. 502.

MACOUN, JOHN.
1898. Report on the "poison-weed" of the Rocky Mountain foothills. Northwest Territories, Department of Agriculture, Bulletin 1, pp. 17–18.

MARIÉ, PAUL.
1885. Recherches sur la structure des renonculacées. Annales des Sciences Naturelles. Botanique, s. 6, t. 20, pp. 5–180, 8 pl.

MARQUIS.
1877. Ueber die Alkaloide des Delphinium staphisagria. Mitgetheilt von Dragendorff. Archiv für experimentelle Pathologie und Pharmakologie, Bd. 7, pp. 55–80.

MEYER, ALBERT.
1885. Ranunculaceen. Wigand, Albert. Botanische Hefte. Marburg. Hefte 1, pp. 3–50, pl. 1.

MILLER, PHILIP.
1760. Figures of the most beautiful, useful, and uncommon plants described in The Gardeners Dictionary. London. v. 2, p. 167, pl. CCL.

NELSON, AVEN.
1896. First report on the flora of Wyoming. Wyoming Agricultural Experiment Station, Bulletin 28, p. 79.

NELSON, S. B.
1906. Feeding wild plants to sheep. Washington Agricultural Experiment Station, Bulletin 73, pp. 4–5, 46–51, 2 fig.

NICANDER.
Theriaca. Line 943.

ORFILA, M. J. B.
1817. General system of toxicology. Abridged and partly translated from the French by Joseph J. Nancrede. Philadelphia. pp. 222–223.
1843. Traité de toxicologie. Paris. ed. 4, t. 2, pp. 120–122.

PAMMEL, L. H.
1910. Manual of poisonous plants. Cedar Rapids, Iowa. [pt. 1], pp. 44, 45, 108, 109.

PLINIUS SECUNDUS, C.
Historia naturalis. Lib. 23, cap. 13. (Bibliotheca Classica Latina, v. 7, 1830.)

VAN PRAAG, J. L.
1854. Delphinin. Toxikologisch-pharmakodynamische Studien. Archiv für pathologische Anatomie und Physiologie [Virchow], Bd. 6, pp. 385–408, 435–457.

RABUTEAU, A.
1874. Contribution a l'étude des effets de la delphine. Comptes Rendus des Séances et Mémoires de la Société de Biologie [Paris], t. 26 (s. 6, t. 1), pp. 286–291.

SCRIBONIUS LARGUS.
De compositionibus medicamentorum. Cap. 166.

SLADE, H. B.
1903. Some conditions of stock poisoning in Idaho. Idaho Agricultural Experiment Station, Bulletin 37, pp. 159–190, 2 pl., 3 fig.

SOLEREDER, HANS.
1908. Systematic anatomy of the dicotyledons. Translated by L. A. Boodle and F. E. Fritsch. Revised by D. H. Scott. 2 v., Oxford.

STRASBURGER, EDUARD; NOLL, FRITZ; SCHENCK, HEINRICH; and KARSTEN, GEORGE.
1908. Text-book of botany. 3d English edition revised with 8th German edition by W. H. Lang. London.

TURNBULL, ALEXANDER.
1838. On the medical properties of the natural order Ranunculaceæ. Philadelphia. pp. 48–53, 72

VESQUE, JULIEN.
1881. De l'anatomie des tissues appliquée a la classification des plantes. Nouvelles Archives du Muséum d'Histoire naturelle, s. 2, t. 4, p. 28.

WATT, GEORGE.
1890. Dictionary of the economic products of India. London and Calcutta. v. 3, pp. 64–71.

WILCOX, E. V.
1897. Larkspur poisoning of sheep. Montana Agricultural Experiment Station, Bulletin 15, pp. 37–51, 3 pl.
1899. Cattle poisoning by the tall larkspur. Montana Agricultural Experiment Station, Bulletin 22, pp. 45–47.

WILLING, T. N.
1900. Northwest Territories, Department of Agriculture, Annual Report, 1899, p. 40.
1900. Noxious weeds and how to destroy them. Northwest Territories, Department of Agriculture, Bulletin 2, p. 27.

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